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A Monthly Journal Devoted to the Art and Science of Surgery

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Vol. 38, No. 5, November, 1955. Surgery is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription price: United States, $14.00 a year; Canada, $14.50; Foreign, $15.50. Entered as Second-Class matter January 20, 1937, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in the U. S. A.

SURGERY

VOL. 38 NOVEMBER, 1955 No. 5

Original Communications

Society for Vascular Surgery

SOME PRACTICAL CONSIDERATIONS IN THE SURGERY OF BLOOD VESSEL GRAFTS

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(From the Department of Surgery, Massachusetts General Hospital)

THE restoration of vascular continuity in the treatment of diseases and injuries of the aorta and major arteries by the use of blood vessel grafts has been the goal of surgeons for many years. The need for this type of therapy has become more evident in recent years because of the increasing incidence of (1) obliterative arterial disease, secondary to atherosclerosis in the aorta and the major arteries to and of the lower limbs, and (2) abdominal aortic arteriosclerotic aneurysms, a fatal disease if untreated. These vascular lesions, which represent degenerative arterial diseases of old age, undoubtedly will continue to increase in number because of the lengthening of the human lifespan, until the etiology of atherosclerosis is better understood and the prophylaxis of this disease is accomplished.

In general, there are five main indications for re-establishing the arterial continuity of the aorta and the major arteries: (1) Obliterative diseases of the aorta and the major arteries of the body. (2) Aneurysms of the aorta and major arteries. (3) Injuries which have resulted in interruption of arterial continuity, secondary to thrombosis or division of an artery. (4) Tumors situated near major arteries in which it is necessary to resect these blood vessels in order to perform an adequate "cancer operation." (5) Congenital vascular lesions.

TYPES OF ARTERIAL LESIONS

Obliterative Diseases.—The most common use of blood vessel grafts is in the treatment of obliterative arterial diseases, involving the aorta and the

Presidential address, read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

arteries of, and those which supply, the lower extremities secondary to atherosclerosis.[4, 15, 19] The chief reason to re-establish arterial continuity in the majority of these patients is to restore the muscular function of the limb by improving the arterial blood supply to it. Intermittent claudication, the most common symptom in these patients, develops because of an inadequate arterial blood supply to the muscles of the lower leg, thigh, or buttocks during muscular exercise, the result of arterial obliteration in the major arteries to the involved limb. The other methods that have been utilized in attempts to relieve these symptoms, including sympathetic ganglionectomy, have little effect on this symptom complex because of the failure to augment sufficiently the arterial blood flow to the muscles. Re-establishment of the vascular continuity in the major arteries to an affected limb restores the blood supply to a nearly normal degree, and results in the restoration of muscular function in the majority of the limbs affected with a return of exercise tolerance to normal in the majority of the patients. In addition to this beneficial effect it will likewise relieve the other symptoms of ischemia, including rest pain. In a few instances it has resulted in the healing of ischemic ulcerations of the lower legs, feet, and toes, thereby saving extremities that otherwise would have had to be amputated.

The arteries most commonly affected by the obliterative process, which can be treated by vascular grafts, are the abdominal aorta distal to the renal arteries, the iliacs, the femoral, and the proximal portion of the popliteal artery. In the case of smaller arteries, such as the posterior tibial, anterior tibial, and peroneal arteries, the lumen of them are so small in the diseased state that vascular anastomosis can rarely be accomplished.

Aneurysms.—One of the most important indications for the use of blood vessel grafts is in the treatment of abdominal arteriosclerotic aortic aneurysms, because of the inevitability of death resulting from rupture of the aneurysmal sac.[1, 5, 20] The duration of life, after the diagnosis of an abdominal aortic aneurysm has been made, is difficult to ascertain because of the impossibility of determining how long the aneurysm was present before it was first discovered. The duration of life after the onset of symptoms, especially that of pain usually referred to the back, can be measured in months, according to most investigators on the subject. For this reason it is believed that the presence of an abdominal aortic aneurysm, whether it is giving symptoms or not, but especially if it is, is sufficient indication to warrant resection of it with replacement of the aorta and its major bifurcation with an aortic graft. In addition, the insertion of a blood vessel graft to restore the arterial continuity after the resection of an aneurysm of the iliac, femoral, and popliteal arteries is the best form of treatment of these lesions. Excision of aneurysms involving these blood vessels is performed chiefly to prevent rupture of the aneurysmal sac, but also, in addition, to relieve pain and to prevent embolization of the arteries distal to the aneurysm from the dislodgment of an intrasaccular thrombus. The results of this method of treatment in this type of lesion have been especially gratifying since the threat of death from rupture of the aneurysm has been removed and at the same time normal arterial circulation

is restored to the parts distal to the site of the aneurysm. The use of arterial homografts is also of great value to re-establish arterial continuity after removal of aneurysms involving other major arteries, including the iliac, femoral, popliteal, subclavian, and carotid arteries.

Vascular Injuries.—The value of vascular grafts in the treatment of major arterial injuries secondary to high velocity missiles has been clearly demonstrated by Jahnke and Seeley[14] and others[12, 21] in reports from the United States Army Medical Department during the recent Korean conflict. Without question, if the more recent developments in this method had been available during the two recent World Wars, a great number of limbs and lives could have been saved. Although these types of injuries occur less frequently in civilian life, there still are a fair number of them. In addition to this type of vascular injury, others are seen secondary to sharp objects or severe contusions, not infrequently associated with fractures that should be treated by this method when the continuity of the artery has been interrupted either by laceration or thrombosis.

Malignant Lesions.—The operability and undoubtedly, in some cases, the curability of malignant lesions that involve major arteries, especially in the neck and limbs, can be greatly extended and improved by utilizing vascular grafts, since much wider excisions of the growths with greater margins of safety will be possible if the involved artery is resected with the tumor and the arterial continuity restored with a graft, thereby preserving the part distal to the tumor.

Congenital Arterial Lesions.—Another important use of vascular grafts is in the treatment of congenital arterial lesions. They are of special value in the infantile type of coarctation of the aorta where the narrowed portion is so long that restoration of the aortic continuity is impossible without the insertion of some type of graft.[10]

TYPES OF GRAFTS

There are three types of blood vessel grafts for practical purposes which are available to restore arterial continuity: (1) the autogenous venous; (2) the homologous venous; and (3) the homologous arterial. A fourth type of graft is a tubular prosthesis made from one of several synthetic fabrics, including Vinyon-N, nylon, Orlon, and Dacron.

The Autogenous Venous Graft.—The chief advantage of this type of graft is that it is tissue obtained from the same individual in whom it is to be implanted, so that there is no antigenic reaction to it and, furthermore, it is a living graft. Another advantage is that it is not necessary to preserve and store the vessel in a blood vessel bank, since it is excised and used at the same time that the grafting procedure is performed. In general, the internal saphenous vein of the thigh is most commonly utilized to replace segments of the femoral and popliteal arteries. The method has a number of disadvantages, however, because, first, in many individuals the vein is not large enough to replace even the femoral or popliteal arteries and, even when it is enlarged,

it is not possible to use it for aortic or iliac grafts. Second, the use of it is also limited because it cannot be obtained in a sufficient length to by-pass some of the extensive areas of obliteration in the arterial system. Third, experience has also shown that veins are not as satisfactory as replacement conduits for blood as the homologous arteries because of a higher incidence of secondary thrombosis immediately after the insertion of them. This, undoubtedly, is the result of a number of factors which, in turn, results in turbulence and stagnation of the blood, which favor thrombosis. They lack the elastic lamina which permits the artery to dilate during systole and contract during diastole, thus acting itself as a pump, so that the blood is kept in motion at all times, thereby probably reducing the incidence of clotting. It is necessary to reverse the vein when it is implanted so that the venous valves will not interfere with the blood flow and, as a result of this change in the direction of it, the smaller end frequently is sutured to the larger end of the arterial tree and the larger venous end to the smaller arterial vessel. This lack of streamlining undoubtedly interferes with the efficiency of the arterial blood flow. The presence of the venous valves and the segmental areas of dilatation of the veins at the site of them may also be a factor in producing turbulence and stagnation with thrombosis. A fourth disadvantage from a technical point of view is that the veins are much more difficult to manipulate and suture, because of their flaccidity, as contrasted with the more rigid walls of the arteries. Despite all these disadvantages, however, the venous graft has been utilized with success in a number of cases and its ready availability, especially for short arterial grafts in the femoral arteries, is to be highly recommended (Table I).

TABLE I. TYPES OF VASCULAR GRAFTS

	NO. GRAFTS	NO. SUCCESSFUL	NO. FAILURES
Autogenous vein	13	8 (61%)	5 (39%)
Homologous vein	9	4 (44%)	5 (56%)
Homologous artery	68	52 (77%)	16 (23%)
Totals	90	64 (71%)	26 (29%)

The Homologous Venous Graft.—This type of graft has the advantage of being obtainable from patients with varicose veins in long segments since the long saphenous trunk in many instances can be removed from the groin to the knee in one piece. It unfortunately has all the disadvantages of an autogenous venous graft, plus the fact that it is homologous tissue which apparently increases the incidence of thrombosis, as shown in our experience (Table I). This type of graft has been most successful for replacement of short segments of the iliac, femoral, and popliteal arteries.

The Homologous Arterial Graft.—At the time of this writing the homologous arterial graft is the most useful and satisfactory of aortic and arterial replacements. One of the chief advantages of this type of graft is that secondary thrombosis occurs less frequently than with other types of grafts. It is possible also to obtain homologous arterial grafts in any desired size or length to replace any of the arteries; thus, an aortic graft can be obtained to replace the aorta and, similarly, it is possible to obtain arteries to replace any of the diseased ones.

The arterial grafts are all obtained at the time of post-mortem examinations. It is recommended that they be removed from the body within six to eight hours after death, although femoral arteries removed as long as thirty-six hours after death have been successfully used. It is important under these circumstances that the body be kept in a refrigerator near 0° C. until the blood vessels are obtained, and that only those from the limbs should be utilized. The proximity of the intestinal tract with its organisms to the abdominal aorta and the iliac arteries with the slow cooling of the trunk may produce changes in these blood vessels when removed later after death so that they are unsuitable for grafts (Table I).

Prostheses.—The synthetic fabrics have proved to be excellent replacement prostheses for large blood vessels such as the aorta and the iliac arteries.[11, 20] Voorhees, Jaretzki, and Blakemore[24] were the first to recommend Vinyon-N cloth, but since then other synthetic fabrics, including Orlon, Dacron, and nylon, have been used with success. The most recent development has been made by Edwards.[8] He has developed the first satisfactory prosthesis to replace short segments of the femoral artery. This type, however, is not suitable for the end-to-side anastomosis which is necessary for suturing grafts to small arteries such as the distal portion of the popliteal artery in the presence of a proximal obstruction. Despite their present imperfections and the fact that homologous arterial grafts are the best available today, it is generally believed that with the aid of modern chemistry the perfect prosthesis will be developed which will replace the natural vascular grafts. The advantages of having available all lengths and sizes of such replacements is almost a utopian dream, inconceivable today to the vascular surgeon, since it would also eliminate the difficulty of procuring and preserving the homografts, and sterilization would also no longer present a problem since the grafts could be autoclaved with the other surgical equipment.

Methods of Preservation.—The utilization of homologous blood vessel grafts has necessitated the development of some method for preserving blood vessels. At the present time there are four methods available which include: (1) the nutrient broth; (2) the quick-freeze; (3) the freeze-dry; and (4) the frozen-irradiated.

1. *The nutrient broth method:* This method was developed by Gross and associates[9] and reported in 1949. It necessitates strict aseptic precautions in obtaining the blood vessel grafts. The blood vessels are then stored in the nutrient broth which is kept at a temperature just above 0° C. The chief disadvantages of this method are the necessity of asepsis in obtaining and preserving them and the fact that the blood vessels can be stored only for a period of approximately thirty to forty days, at which time they must be discarded because of the degenerative changes that take place in them which make it inadvisable to use them. This method has been replaced in most major vascular centers by either the freeze-dry or the frozen-irradiated technique.

2. *The quick-freeze method:* This is the most simple,[6] but unfortunately not the most satisfactory method of preservation because the grafts must be

obtained by the aseptic technique. The vessels, after they have been removed, are washed with saline solution, sealed in a glass or plastic container, and immediately stored in this manner in carbon dioxide ice or in a refrigerator at –60° C. until they are to be used. The chief disadvantages of this method are (1) the difficulty in obtaining the blood vessels under aseptic conditions in the autopsy room and (2) the maintenance of them at a constant low temperature.

3. *The freeze-dry method:* This method has been developed by Hyatt,[13] Pate,[18] and Brown.[2] The blood vessels do not need to be taken under aseptic precautions with this technique since they are sterilized in liquid ethyl dioxide or according to Szilagyi[22] with *B. propiolactone* for a short period of time after their removal. Then they are frozen in a carbon dioxide–alcohol mixture and maintained at this lower temperature while they are completely dehydrated in a vacuum of approximately 0.025 mm. of mercury. After a period of freeze-drying for from eight to twelve hours, the vessels are sealed in glass tubes. The greatest advantage of this method is that after this preparation the blood vessels may be kept at room temperature and may be transported in the glass tube containers without any special precautions as to temperature control.[7]

4. *The frozen-irradiated method:* This method was first described by Meeker and Gross[17] in 1951, and is an outgrowth of studies on the effect of irradiation of biologic material by high energy roentgen and cathode rays carried out in 1948 by Trump and Van de Graaff.[23] Meeker and Gross utilizing this method, have shown that it is possible to sterilize contaminated aortic blood vessel grafts by exposing them to the electron beam of a Van de Graaff cathode ray machine of 3,000,000 volts for periods of relatively few seconds. They also showed that if the correct dosage and exposure time are used, there is very little injury to the blood vessel wall, including the intimal lining. At the present time the method utilized in our clinic is to remove the aorta and the major blood vessels of the pelvis and thigh down to the popliteal bifurcation. These are divided into segments of various lengths, they are sealed in two polythene bags, one within the other, and then quickly frozen at a temperature of –65° to 70° C. After a sufficient number of grafts has been obtained they are taken to the Massachusetts Institute of Technology in Cambridge, Massachusetts, and sterilized under the 3,000,000 volt cathode ray machine, as described by Meeker and Gross. They are transported to and from the Institute in a vacuum flask containing dry ice to maintain a low temperature and, after sterilization, they are replaced in the low temperature refrigerator and stored until ready for use at a temperature of –65° to 70° C.

A notebook is kept to record the data for each graft, including the time after death it was obtained, the age of the donor, the length and caliber of the vessels, and when and by whom it was sterilized. It has been learned, however, that it is unsafe to rely solely on this record, in case of human error; therefore, an automatic check of the sterility of the graft has been devised. A small, clear glass bead is placed in the outer polythene bag before sealing it. This turns a deep-brown color as a result of exposure to the cathode rays used for sterilizing the grafts. It is possible by noting this color change to have an

automatic record that the graft has been sterilized. This check on the sterility is of extreme importance because both sterilized and unsterilized grafts are kept in the same refrigerator and it is impossible to ascertain by inspection of the graft whether it has been sterilized. If an unsterile one is used it will result in the loss of the limb and perhaps the life of the patient, due to the sepsis and arterial hemorrhage usually at the proximal anastomosis.

The advantages of this method are that blood vessels do not need to be removed under aseptic conditions. They may also be obtained from patients who have died from sepsis or malignant disease because the cathode rays kill both bacteria and tumor cells. They can be sterilized in any size or length that is desired, and as long as the refrigerator is maintained at approximately -60° C. they may be kept for indefinite periods of time. It is believed also that this method may damage the blood vessel less than any of the other methods of preservation, and for that reason smaller-calibered blood vessels may be used for the grafting procedures. As far as is known at this time, this is the only method that has made it possible to utilize blood vessel grafts extending from the abdominal aorta just distal to the renal arteries, down to the popliteal arteries.

Arteriography.—In order to be able to restore satisfactorily the arterial circulation, especially in cases of obliterative arterial disease, by means of blood vessel grafts, it is essential that an exact picture of the extent and nature of the arterial lesion be obtained. For this reason it is necessary to resort to aortography and arteriography by injecting into the aorta or artery a radiopaque solution of one of the organic iodine compounds commonly used for intravenous pyelography. In our clinic a 70 per cent solution of Diodrast is used for aortography using approximately 50 c.c. injected through a large-bored needle that has been inserted into the aortic lumen through the translumbar route. It is preferable to get the point of the needle above the renal arteries so that the relation of these vessels to the diseased aorta can be determined. It is also possible with aortography to visualize the common, external, and internal iliac arteries, and, with the Menendez technique, the femoral arteries also may be demonstrated. For arteriography of the femoral arteries alone, a 35 per cent solution of Diodrast is injected percutaneously into the common femoral artery in the groin. Both aortography and femoral arteriography may be performed under local anesthesia, but in some cases anesthesia with Pentothal sodium is necessary to obtain satisfactory aortograms.

Requirements for a Successful Blood Vessel Graft.—The success of any blood vessel grafting procedure is dependent upon a number of factors. Attention to all of them is of extreme importance because the failure of a graft to function may result in more ischemia to the limb with danger of gangrene, in addition, in many instances, to putting a critically ill patient through a useless operative procedure. The following eight requirements, it is believed, are essential for a successful vascular graft. (1) A suitable graft must be available to by-pass the obstructed portion of the diseased blood

vessel. This obviously is of prime importance to obtain a successful result. It necessitates the establishment of a blood vessel bank in which all the various types of arterial grafts necessary for this type of surgery are preserved and ready for use. These should include the abdominal aorta with its bifurcation and the attached iliac arteries, various lengths of iliac, femoral, and popliteal arteries, and some of the smaller ones such as the brachial artery. It is important to have them of different calibers so that the correct size can be utilized in a given patient because of the variation in the caliber of the same artery in different individuals. (2) The site of the proximal anastomosis must be at a level sufficiently high on the patient's arterial system to supply an adequate inflow of arterial blood through the graft. It should be of sufficient volume and under great enough pressure to maintain an adequate rate of blood flow in the graft to prevent thrombosis of it. In some cases, to fulfill this requirement, it may even be necessary to replace the abdominal aorta, as well as the iliac, femoral, and popliteal arteries. (3) An equally important requirement is that there must be an adequate arterial bed distal to the distal anastomosis of the graft to permit a satisfactory arterial outflow; otherwise there may be stagnation and interference of blood flow through the graft with resulting thrombosis. A preoperative arteriogram as a rule will give this information. (4) A patient should not be operated upon until satisfactory arteriograms are obtained to satisfy especially the second and third requirements so that the correct site of the proximal anastomosis may be selected to obtain an adequate arterial inflow and, at the same time, to determine if there are sufficient arteries distal to the occluded vessels to carry the blood away from the graft. (5) The surgeon must develop a meticulous technique, avoiding unnecessary trauma to the host arteries and the graft itself. The anastomosis must be performed with delicate instruments and fine atraumatic suture materials. (6) The next requirement is the use of the anticoagulant heparin during the operative procedure. It is recommended that it should not be used postoperatively because severe secondary bleeding in the operative wounds too frequently develops. It is injected into the host arteries proximal and distal to the occluding clamps to prevent thrombosis from occurring in them while the anastomoses are being performed. If this precaution is not taken, thrombi are very likely to form, especially distal to the distal anastomosis, so that when the blood is allowed to flow through the graft the blood clot, if present, will be carried distalward to embolize the arterial tree with danger of gangrene and the loss of the limb. Experience has demonstrated that a relatively large amount of a weak solution of heparin is better than a smaller quantity of a more concentrated solution, because the arterial tree should be heparinized for a considerable distance beyond the distal occluding clamp since thrombosis resulting from cutting off the arterial blood flow is most likely to occur just beyond the occluding clamp. It may also originate at any of a number of the atheromatous plaques some distance from the point of occlusion. It is believed advisable also to inject some of the same solution proximal to the proximal occluding clamp. For femoral grafting procedures about 10 to 20 c.c. of a 0.01 per cent solution of heparin is injected both proximally and

distally into the femoral and popliteal arteries at the site of the proximal and distal anastomoses. For abdominal aortic grafts 20 to 30 c.c. of the same solution is injected into the iliac arteries as soon as they have been clamped and divided and 5 to 10 c.c. into the aorta above the occluding clamp. At this latter site, or if the proximal anastomosis for a femoral graft is an iliac or a common femoral artery, the heparin is best injected through a small caliber (No. 24) needle. At the other sites it is injected through a small polythene tube, attached to a syringe, which is inserted through the open end of the artery or the arteriotomy incision and between the jaws of the bulldog clamp used to occlude the artery. (7) Another requirement is the meticulous apposition of the surrounding tissues of the host to the graft and the elimination of all dead spaces in which serum and blood clot might collect between them. This is important since all homologous blood vessel grafts are dead tissue and the survival of them depends upon their replacement by the host tissues which necessitates close approximation of them and the graft. (8) Next is the employment of the most rigid aseptic precautions possible, and at the same time it is believed imperative that the antibiotics, usually penicillin and streptomycin, be administered both preoperatively and postoperatively in order to prevent infection in the surgical wound. If it occurs, necrosis of the graft develops with secondary rupture, necessitating ligature of the arterial supply proximal to the proximal anastomosis; in all the cases in which this has been performed the result has been the loss of the limb.

The Causes of Failure of the Graft to Function.—There are a number of causes for the failure of a graft to function. The following are some of the more important ones which should be noted since they are preventable. The most common is poor surgical technique with stenosis or damage to the intima of the host artery, especially at the site of anastomosis. This may result in the slowing of the blood flow with the development of a thrombosis at either the site of the proximal or distal anastomosis. It is of utmost importance also, for the same reason, to obtain an intima-to-intima approximation of the two arteries, especially in the smaller blood vessel anastomoses, such as in the femoral and popliteal arteries. An everting mattress suture should be utilized to accomplish this, especially in the end-to-end type of anastomosis. In some instances, failure is believed to have occurred because of the stenosis at the sites of anastomoses, especially with the end-to-end type, when used for femoral and popliteal arteries. Another cause has been that the upper anastomosis was not placed proximal enough on the host's artery to obtain an adequate delivery of blood to the graft.

In others, failure has resulted because the arterial system distal to the graft has not been of sufficient caliber to permit an adequate outflow. At the present time, we have discontinued the end-to-end type of anastomosis for the smaller arteries and are utilizing the end-to-side type[3] which enables one to make a much wider anastomotic stoma, and also to place the proximal anastomosis as high on the common femoral or external iliac arteries as necessary to get an adequate source of arterial blood. This method, in addition, preserves all the anastomotic branches distal to the proximal anastomosis so that if the

graft becomes occluded from thrombosis, the host arteries will still continue to function. Infection with secondary hemorrhage is another cause of failure which fortunately rarely occurs if strict asepsis is maintained along with adequate antibiotic therapy. Leakage from an anastomosis line, twenty-four to forty-eight hours after the graft has been inserted, invariably means the presence of infection and demands the immediate removal of the graft to prevent exsanguinating hemorrhage at a later date.

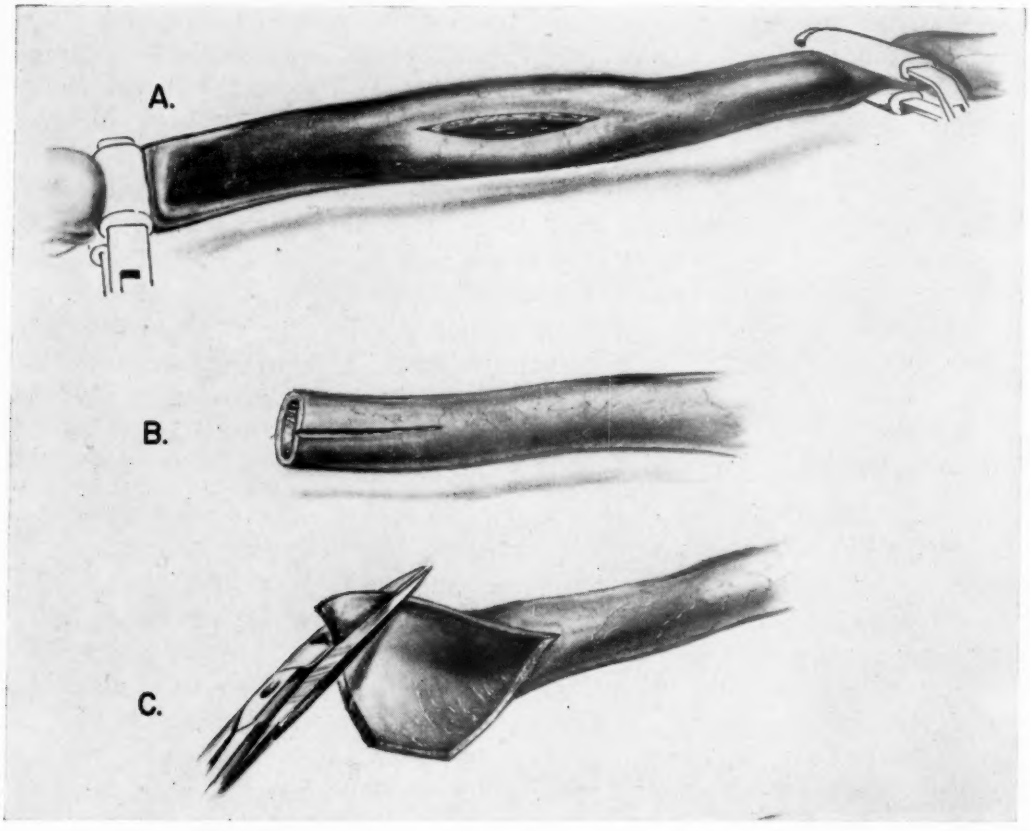

Fig. 1.—An artist's drawings demonstrating the end-to-side technique of an arterial anastomosis. *A*, A portion of the host artery is isolated between two bulldog clamps, in which a longitudinal incision is made approximately two to three times as long as the diameter of the artery.

B, The graft, either venous or arterial, is prepared by making a longitudinal incision from the cleanly divided end of it, approximately as long as the incision in the host artery.

C, The corners of the arterial flap are cut away.

Failure of the graft may also result from a secondary thrombosis which has formed in the host arteries during the operative procedure because of the failure to use heparin or a sufficient amount of it in the arteries proximal and distal to the occluding clamps. This complication may result in embolization of the artery distal to the graft from such a clot when the anastomoses are completed and the clamps are removed. Failure may also result from the utilization of a poor graft, especially one that is too small in caliber to carry a sufficient volume of blood through it.

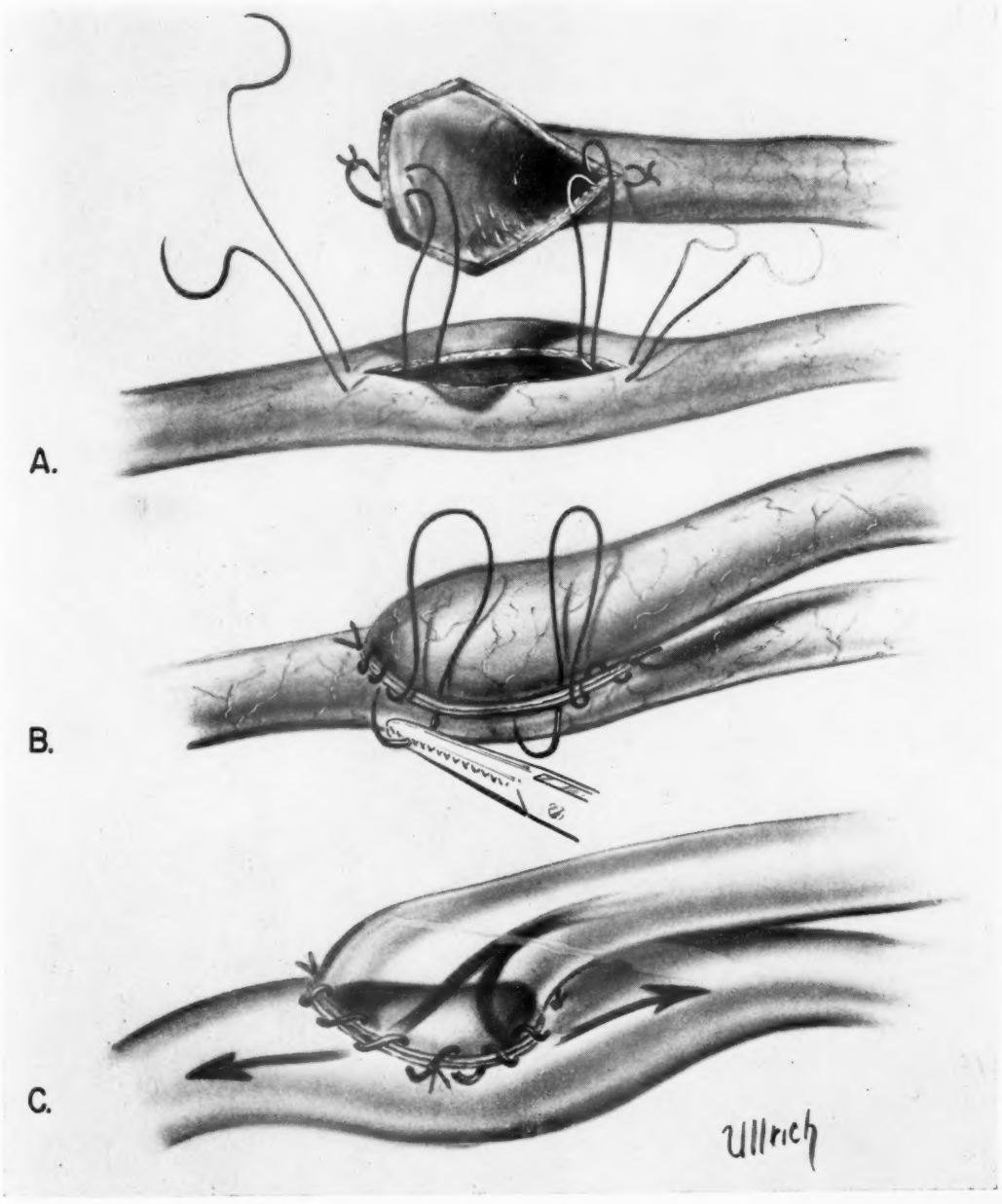

Fig. 2.—The end-to-side technique (cont'd): *A*, Two mattress sutures with needles at each end of 5-0 silk are first used to join the graft with the host artery as shown. The needles are passed through the graft wall from outside to inside and on the host artery in the reverse direction in order to prevent dislodgment or separation of the friable intima of the host artery.

B, The mattress sutures are tied and then each end is used to complete the anastomosis with a running over-and-over type of stitch. The two ends are tied in the middle of the suture line. Note again that the needles always pass from the inside to the outside of the host artery, an important step to prevent separation and fragmentation of the intima.

C, The completed anastomosis. The arrows demonstrate the flow of blood in both directions in the host artery. Note also the funneling or widening produced in the end of the graft and at the site of the anastomosis in the host artery, an important factor in reducing the incidence of thrombosis at the anastomosis site.

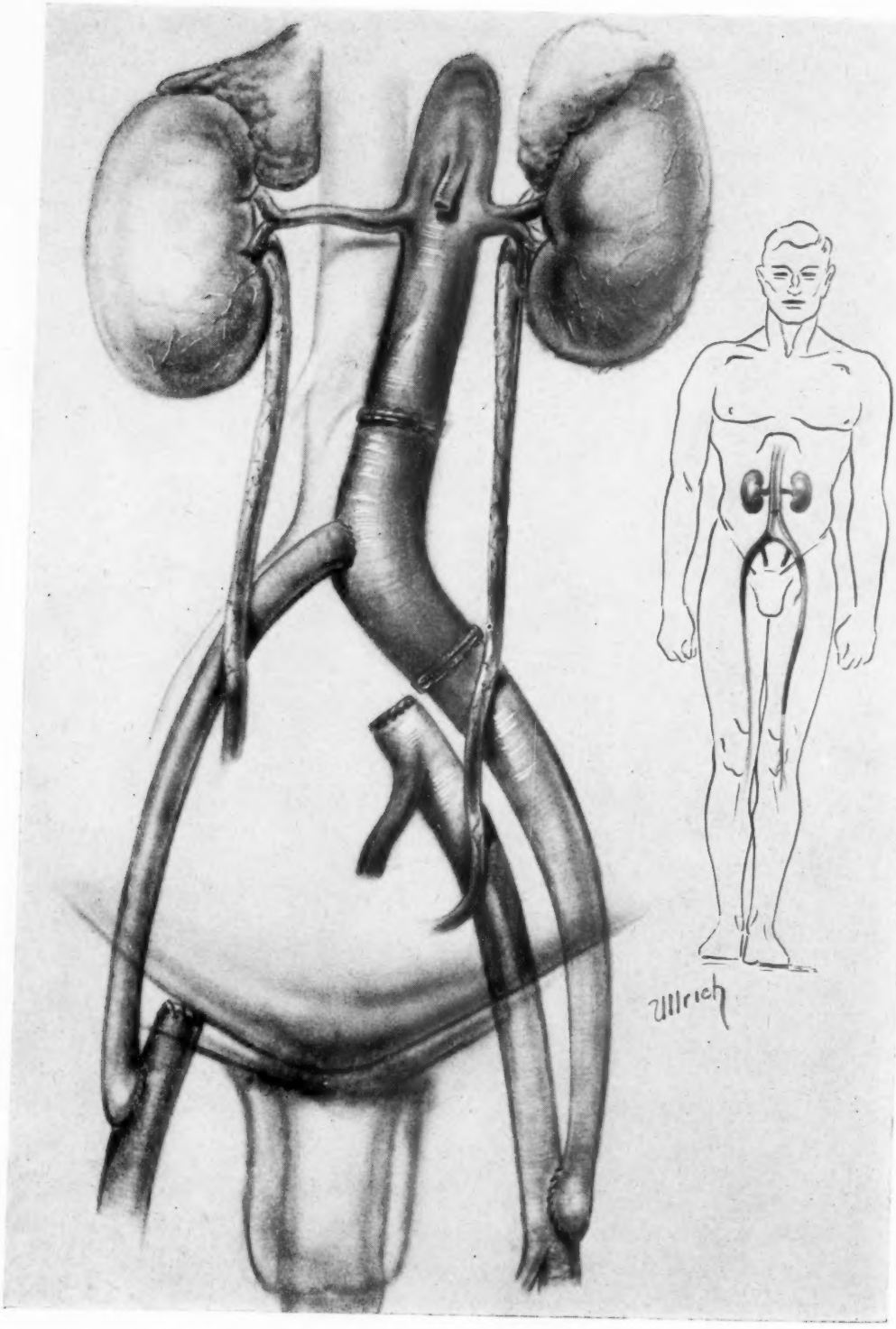

Fig. 3.—*(For legend see facing page.)*

Types of Anastomoses.—For practical purposes there are two methods for anastomosing venous or arterial autografts or homografts to the host arteries or to other grafts. They are performed either by (1) the end-to-end or (2) the end-to-side technique. The latter method, first described by Kunlin[16] for venous autografts and later modified by Cockett[3] for arterial homografts, is more easily constructed and has the additional advantage of producing an anastomotic stoma which is wider than the caliber of either the graft or the host artery (Figs. 1 and 2) so that thrombosis at the site with failure of the graft is much less likely to occur than when the end-to-end type of anastomosis is used, since the latter invariably produces a narrowing of the arterial lumen. The end-to-side technique has also permitted the anastomosis of arterial grafts to extremely small and diseased arteries which would have been impossible with the end-to-end method; because of the poor streamlining with the end-to-side method resulting, perhaps, in more turbulence of the blood stream, it was feared that there would be an increased tendency to thrombosis, but this has not been true; in fact, it has been just the opposite.

The end-to-end type of anastomosis is most useful for the proximal suture of an aortic graft to the aorta or common iliac limbs of an aortic bifurcation graft to the common iliac arteries of the host or another graft. The utilization of both these methods of anastomoses is well demonstrated in Fig. 3.

The end-to-side method has another advantage in that the host artery does not have to be severed, thus preserving the functioning of the collateral channels whether the graft remains patent or becomes occluded. Of utmost significance also, it permits the location of the proximal anastomosis at a site where there is an adequate blood flow, without jeopardizing the existing circulation, so that very long occluded segments of the femoral artery may be by-passed by constructing an end-to-side anastomosis to either the common femoral or external iliac artery and the distal one by the same technique to the distal portion of the popliteal artery. Still another advantage of this technique is that it is not necessary to excise any of the diseased artery of the host, thus avoiding operative trauma and especially injury to the concomitant vein which, if it occurred, might result in deep venous thrombosis and pulmonary embolism.

The technique of the end-to-side type of anastomosis has also been utilized to construct end-to-end anastomoses, especially in suturing two grafts together (Fig. 4). In such cases the ends of each graft are prepared in a manner similar to that of the graft for an end-to-side anastomosis. The end

Fig. 3.—A artist's drawing demonstrating the use of the end-to-side type of anastomoses for the implanting of extensive grafts for aortic and iliac obliterative arteriosclerotic disease. The patient, F. B. No. 800230, a 54-year-old man had had the right common iliac artery resected and arterial continuity restored with a saphenous vein autograft in 1953. This functioned for one year, then occluded. At the second operation on Jan. 21, 1955, the aorta was found badly diseased so it was resected. Arterial continuity was restored to the left lower extremity by utilizing homograft segments of a thoracic aorta and a large femoral artery. The proximal anastomoses were the end-to-end type, the distal one to the left common femoral artery was by the end-to-side technique, and were performed at this level because of a nearly obstructed left external iliac artery. A large femoral homograft was used to restore the arterial continuity of the right lower limb by end-to-side anastomoses as shown. These grafts were sutured together in this manner because a good aortic bifurcation homograft was not available. The circulation and pulses were restored to both limbs and they been maintained satisfactorily to date. A further period of follow-up is necessary, but after approximately six months the result can be classified as excellent.

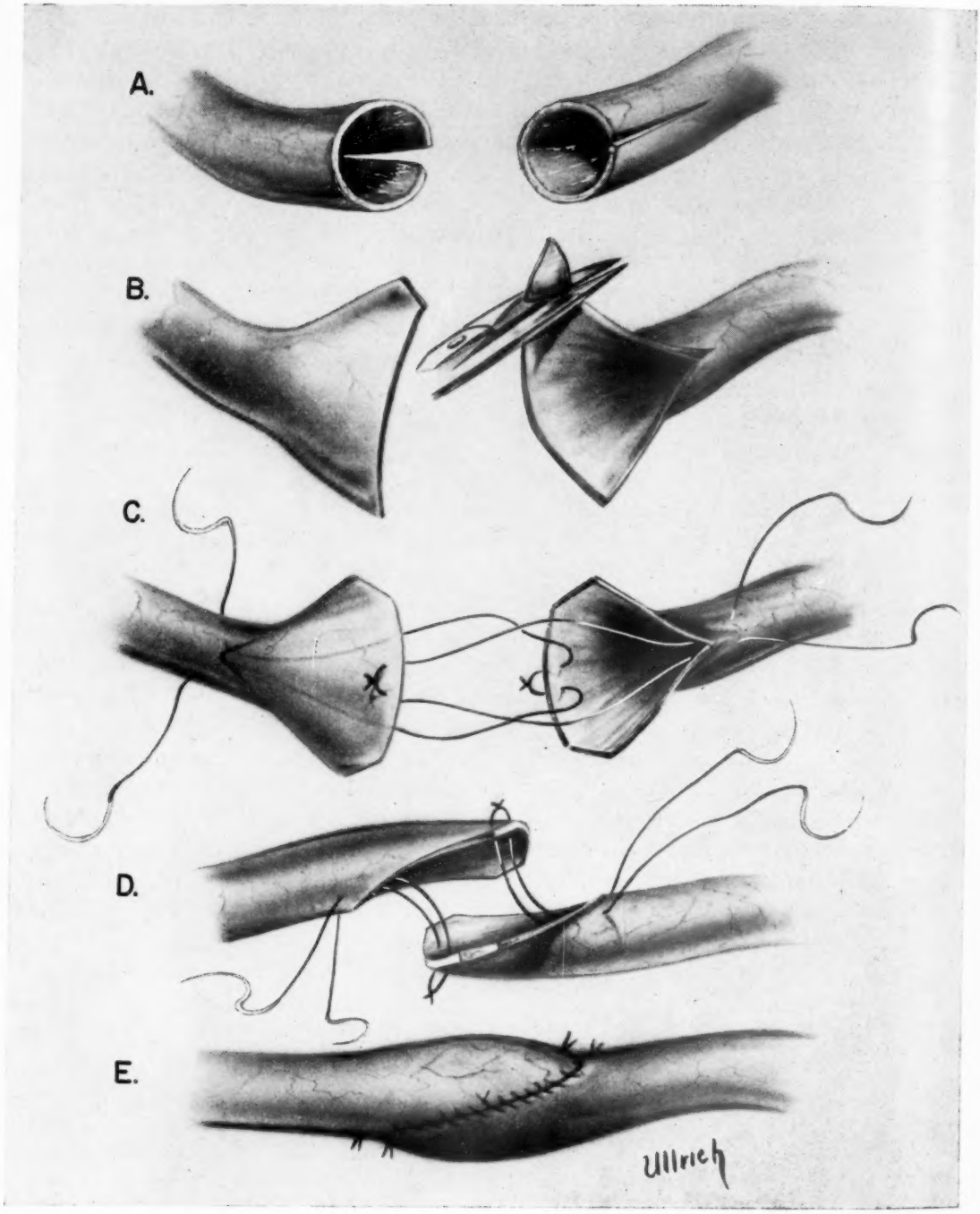

Fig. 4.—An artist's drawings demonstrating an end-to-end anastomosis by the end-to-side technique. *A*, The cleanly divided ends of the grafts are incised longitudinally about two to three times the length of their diameters.

B, The corners of the square-ended arterial flaps are trimmed.

C and *D*, Two everting mattress sutures of 5-0 silk are first placed to bring together the middle of each arterial flap of one graft to the acute angle of the longitudinal incision of the other graft.

E, After tying the mattress sutures the ends are used to complete the anastomosis using a running over-and-over type of stitch, which produces excellent intima-to-intima approximation.

of each vessel is sutured to the apex of the longitudinal cut in the other by a mattress suture. The anastomosis is then completed by suturing each side with a running over and over suture of fine 5-0 silk. If the longitudinal incisions in the vessels are made twice as long as the diameter of them, the completed anastomosis results in a beautifully streamlined union, without any stenosis. This method has proved of great value when a short segment of a saphenous vein autograft or another arterial homograft has had to be utilized to lengthen a homograft which was too short to bridge the distance between an adequate proximal arterial supply and a good distal artery.

TABLE II. COMPARISON OF FROZEN IRRADIATED FEMORAL ARTERIAL HOMOGRAFTS ([A] END-TO-END ANASTOMOSES; [B] END-TO-SIDE ANASTOMOSES)

	END-TO-END	END-TO-SIDE
Number of grafts	22	16
Ages of patients	46 to 81 yr.	42 to 74 yr.
Mean age	60 yr.	59 yr.
Length of grafts	15 to 60 cm.	23 to 60 cm.
Mean length	34 cm.	42 cm.
Number of immediate failures	5 (23%)	1 (6%)
Number of early failures	2 (9%)	0
Number of late failures	9 (41%)*	2 (12%)†

*Failures due to thrombosis of grafts.
†Failures due to sepsis—grafts patent.

The incidence of immediate, early, and late failures secondary to thrombosis in most instances has been greatly reduced for femoral arterial homografts by utilizing the end-to-side technique of anastomosis. In a group of twenty-two homografts of this type implanted with the conventional end-to-end type of anastomosis, there were five (23 per cent) immediate, two (9 per cent) early, and 9 (41 per cent) late failures, whereas in a similar group of sixteen femoral arterial homografts using the end-to-side technique there were only one (6 per cent) immediate, no early, and 2 (12 per cent) late failures; and it is of significance that these two late failures were the result of rupture of the grafts secondary to sepsis and not due to thrombosis (Table II). This superiority of the end-to-side technique is also demonstrated in an analysis of immediate thromboses occurring in common iliac, external iliac, femoral, and popliteal anastomoses; thus in a group of sixty-six end-to-end anastomoses there were 16 or 24 per cent that thrombosed immediately, whereas in fifty-eight of the end-to-side type there were only 2 or 3 per cent that thrombosed (Table III).

TABLE III. COMPARISON OF IMMEDIATE THROMBOSIS WITH (A) END-TO-END ANASTOMOSES (B) END-TO-SIDE ANASTOMOSES FROZEN IRRADIATED ARTERIAL HOMOGRAFTS

	END-TO-END		END-TO-SIDE	
ARTERY	TOTAL NO.	NO. THROMBOSIS	TOTAL NO.	NO. THROMBOSIS
Common iliac	14	4 (29%)	1	0
External iliac	7	1 (14%)	27	1 (4%)
Femoral	22	5 (23%)	12	0
Popliteal	23	6 (26%)	18	1 (6%)
Total	66	16 (24%)	58	2 (3%)

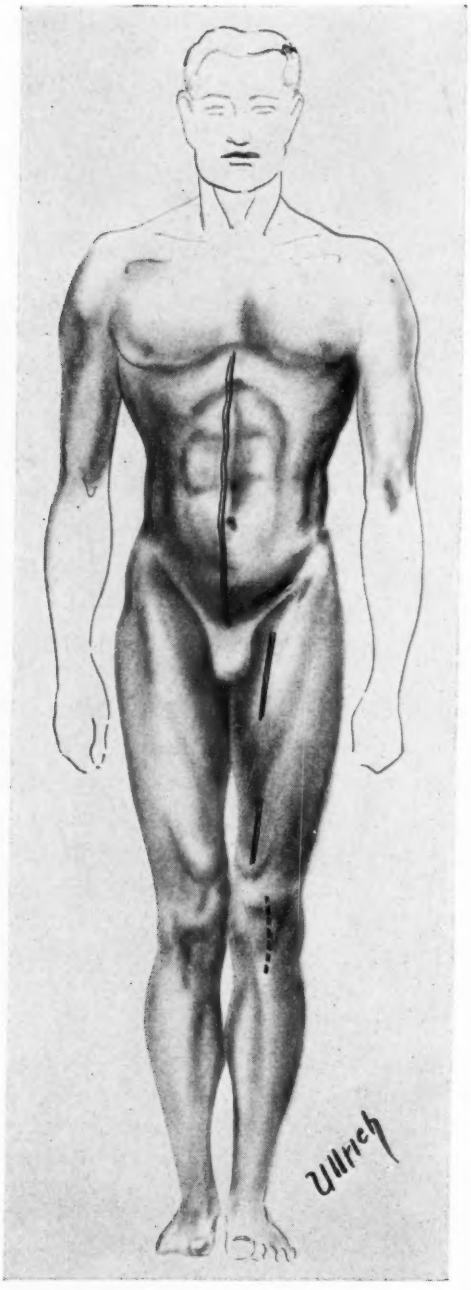

Fig. 5.—An artist's drawing demonstrating the location and extent of the abdominal incision for aortic bifurcation grafting procedures; note that it is a right paramedian type. The multiple incisions used for long femoral grafting procedures are also shown, including an upper oblique one above the crease in the groin for the proximal anastomosis to the common femoral artery and the lower one for the distal anastomosis to the popliteal artery. For these extensive femoral grafting procedures the latter incision is made first with the patient prone, to make certain a graft can be anastomosed to the popliteal artery. After this anastomosis is completed the patient is placed supine, the other incisions in the thigh are made to tunnel the graft up to the groin and the proximal one to perform the anastomosis between the graft and the common femoral artery. It has been found these multiple incisions heal better than one long one.

Incisions.—Adequate exposure is a "sine qua non" for this type of surgery. The insertion of an aortic graft necessitates an incision extending from the xiphoid process to the pubis (Fig. 5) and, in a few instances, it has been necessary to sever the costal arch in order to get adequate proximal exposure and control of the aorta above the renal arteries. At the present time the routine is to use a right paramedian incision since this allows a turning out of the entire small intestinal tract with less danger of damaging its mesentery than if a left abdominal incision is used. Oblique incisions in the thigh are utilized for the femoral and popliteal arterial grafts. It is recommended that multiple incisions in the lower extremity be used rather than making one long incision from the inguinal ligament to the popliteal space for a long grafting procedure. It is readily possible to tunnel beneath the sartorius muscle between the upper and lower thigh incisions for the placement of a long femoral arterial graft extending from the common femoral artery to the popliteal space. The incisions should be closed primarily without drainage. This, of course, necessitates careful and meticulous hemostasis before the closure is performed. Adequate exposure of the artery proximal and distal to the diseased portion of it should be obtained, utilizing incisions which lie over and parallel to them. If necessary, natural skin creases, such as in the groin and the popliteal space, are cut across. Careful closure of the incisions will prevent contractures in these regions. In the more recent extensive femoral arterial grafts when the proximal anastomosis is either to the common femoral or the external iliac artery, the exposure of these vessels is through an oblique incision just above the groin crease. This has the advantage of permitting a better approximation of the host tissues to the graft during the closure of the incision (Fig. 5).

CONCLUSIONS

It is believed at this time that the use of homologous arterial grafts is a method of treating aortic and major arterial lesions which supplants all others in many patients. It is a feasible procedure today because of better surgical equipment and suture material; the availability of the anticoagulant heparin to prevent the clotting of the blood during the operative procedure and the antibiotics to prevent infection; improved anesthesia techniques, including the catheter spinal type of anesthesia which has been most commonly used; the better preoperative and postoperative care of these critically ill patients; and last and perhaps most important of all is the fact that we now have arterial homografts available in our blood vessel bank for the performance of these highly technical procedures. The end-to-side type of vascular anastomosis has made it possible to extend the homografting procedures to include cases that could not possibly be accomplished with the end-to-end method. The chief contraindications to this type of surgery are (1) severe coronary disease which would make these long operative procedures too dangerous to perform, (2) extensive obliterative arterial disease involving the aorta and the major arteries so that the grafts can not be anastomosed satisfactorily to the host arteries, and (3) obliteration of the popliteal artery and its terminal branches.

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EXPERIMENTAL AND CLINICAL OBSERVATIONS OF NEUROGENIC FACTORS IN CORONARY ARTERY DISEASE

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ANGINA caused by diseases that reduce coronary blood flow foreshadows a future fraught with recurrent pectoral pain, myocardial necrosis, diminished cardiac reserve, or disabling fear. Psychotherapy, selected medicaments, and increased leisure attempt to prevent or delay these developments. Some reliance is put upon naturally developing collateral channels but many surgical methods have been devised to replenish the vascular supply of the ischemic heart.

Surgical attempts to relieve this process have been directed along three lines: (1) to revascularize the heart by adding to the ischemic myocardium a new blood supply from some exogenous source[1-7]; (2) to lower the general body metabolism and thus reduce the amount of work the heart is required to do[8]; (3) interruption of the sympathetic nervous system to the heart for the relief of angina.[9]

This study is concerned with investigating neurogenic factors which influence coronary circulation. Clinical observations strongly imply that such factors are important, not only in precipitating angina in an irritable or damaged coronary vascular bed, but also in precipitating myocardial infarction.[10-12] This suggests that coronary vasoconstrictive impulses which influence the coronary circulation must be considered in the treatment of coronary insufficiency. We have investigated the role of certain neurogenic influences on the coronary circulation in the dog and have applied these findings to human beings with coronary artery disease.[13, 14]

In an attempt to investigate the role of neurogenic factors in coronary disease, twenty-four dogs were subjected to vagal and sympathetic stimulation. Each was anesthetized with Nembutal and aerated with intermittent positive pressure. Preliminary electrocardiographic tracings were taken in the limb leads for a base-line study. A thoracotomy incision was made through the left fourth intercostal space. The lung was retracted posteriorly, exposing the mediastinum. The vagus nerve was identified and exposed from the thoracic inlet to the diaphragm. All branches of the vagus and the recurrent laryngeal nerve were preserved. Control electrocardiographic tracings were again made after the vagal dissection. The animals were then ready for selective stimulation of the vagus nerve and its branches.

Stimulation was by induced (faradic) current. The vagus trunk was stimulated at three points along its course through the thoracic cavity, at the thoracic inlet, at the level of the hilus, and at a level below the hilus. Stimulation at each of these sites variably initiated sinus auricular standstill, auricular

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

fibrillation, nodal tachycardia, and ventricular extrasystoles. Similar changes in rhythm resulted from stimulation of the recurrent laryngeal nerve. On one occasion, stimulation of the vagus at the thoracic inlet produced T-wave inversion.

Fibers contributed to the cardiac plexus by the vagus nerve and its recurrent branch were next studied. Fibers of the vagus trunk were identified in the mediastinum, medial to the main vagal bundle (Fig. 1). The branches from the recurrent laryngeal nerve were located in the sulcus between the aorta and

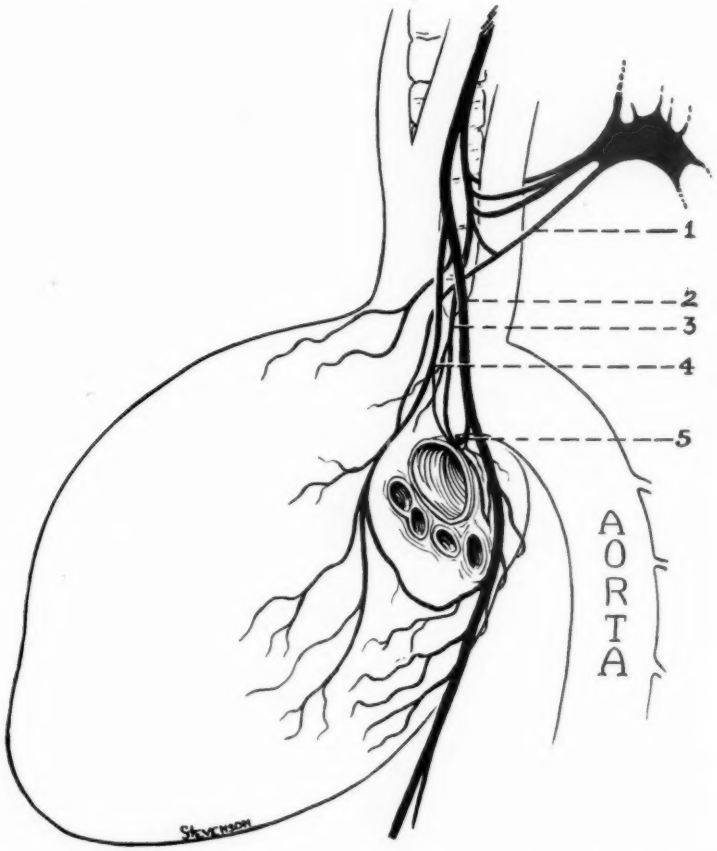

Fig. 1.—A composite drawing of several vagal dissections in the dog. *1*, Sympathetic branch from the stellate ganglion; *2*, left main vagal trunk; *3*, branch of vagus containing probable fibers from the stellate ganglion; *4*, main cardiac nerve from the vagus (there may be more than one branch); *5*, recurrent laryngeal branch of the vagus nerve and fibers that pass to the cardiac plexus (not shown).

the pulmonary artery lateral to the ligamentum arteriosum. Stimulation of the vagal branches produced striking and hitherto unreported electrocardiographic changes which could not be duplicated by stimulating the vagus at any other area. The responses consisted not only of arrhythmias of the sort obtained by stimulating the vagus at other areas, but produced effects which followed a pattern similar to that of acute myocardial infarction in human beings. In those leads where the T waves were initially upright, the T waves became higher and sharper; the ST segments became elevated and coved. Subsequently, the

T waves tended to invert as the ST segments descended toward the base line. Later, the T waves became symmetrically inverted. These changes were produced by stimulation of from 0.1 to 1.0 second's duration and persisted for as long as two to three minutes. Gradually the electrocardiographic tracing returned to normal (Figs. 2 and 3).

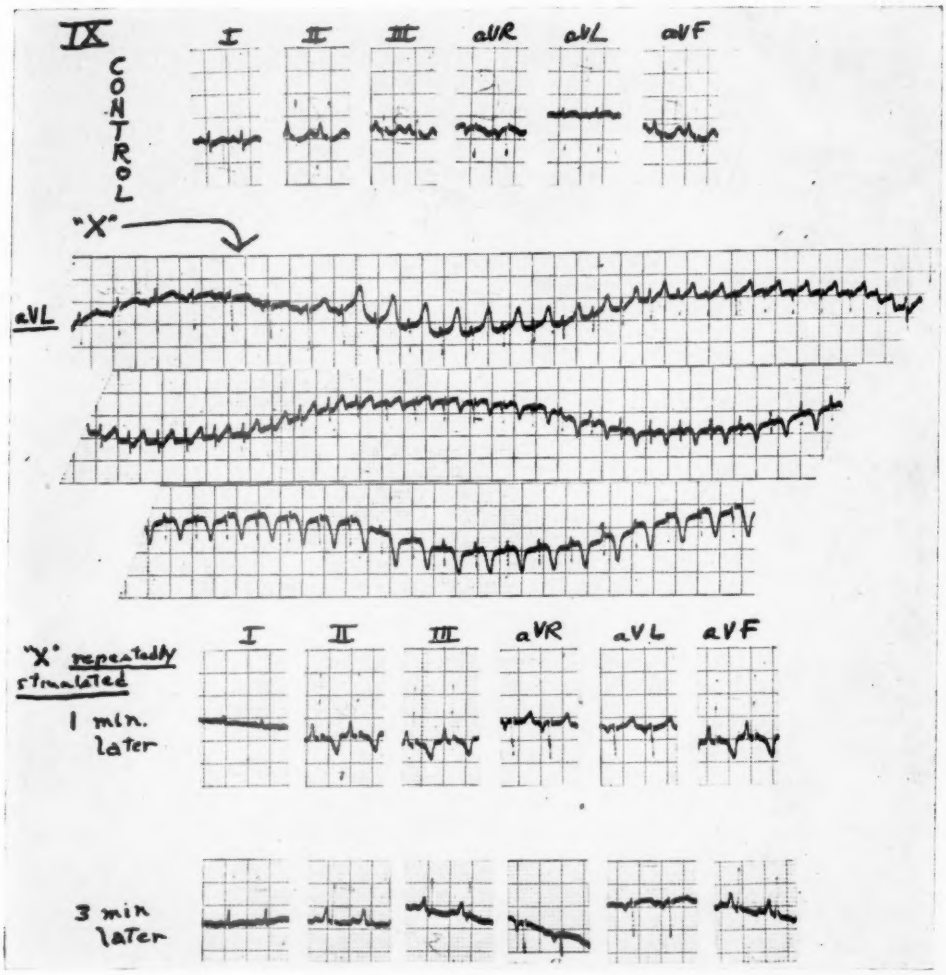

Fig. 2.—Effect of stimulating vagal cardiac branches in Dog No. 9. Control studies in various leads before stimulation, one minute, and three minutes after stimulation. Arrow indicates point of stimulation. This was followed by a slight increase in heart rate, progressive increase in height of the T wave, then lowering and finally symmetrical inversion of the T wave (Lead aVL). One minute after repeated stimulation (*x*), the record shows T waves inverted where originally upright, and upright where initially inverted. Three minutes later the record is returning toward the control appearance.

These branches of the vagus were then cut and the proximal and distal ends separately stimulated. Stimulation of the proximal ends produced no effect. Stimulation of the distal ends produced the ST and T changes as described.

Since stimulation of certain branches of the vagus produced a different effect than that obtained by stimulating the main vagus trunk, it was thought

that perhaps other fibers not vagal in origin might contribute to the effect obtained. The stellate ganglion was isolated and stimulated while intact. Its branches were then stimulated proximally and distally after being severed. Stimulation of the stellate ganglion and the distal cut end of this ganglion produced characteristic changes which were unlike those obtained by stimulating the branches of the vagal cardiac nerves. These changes consisted in an increase

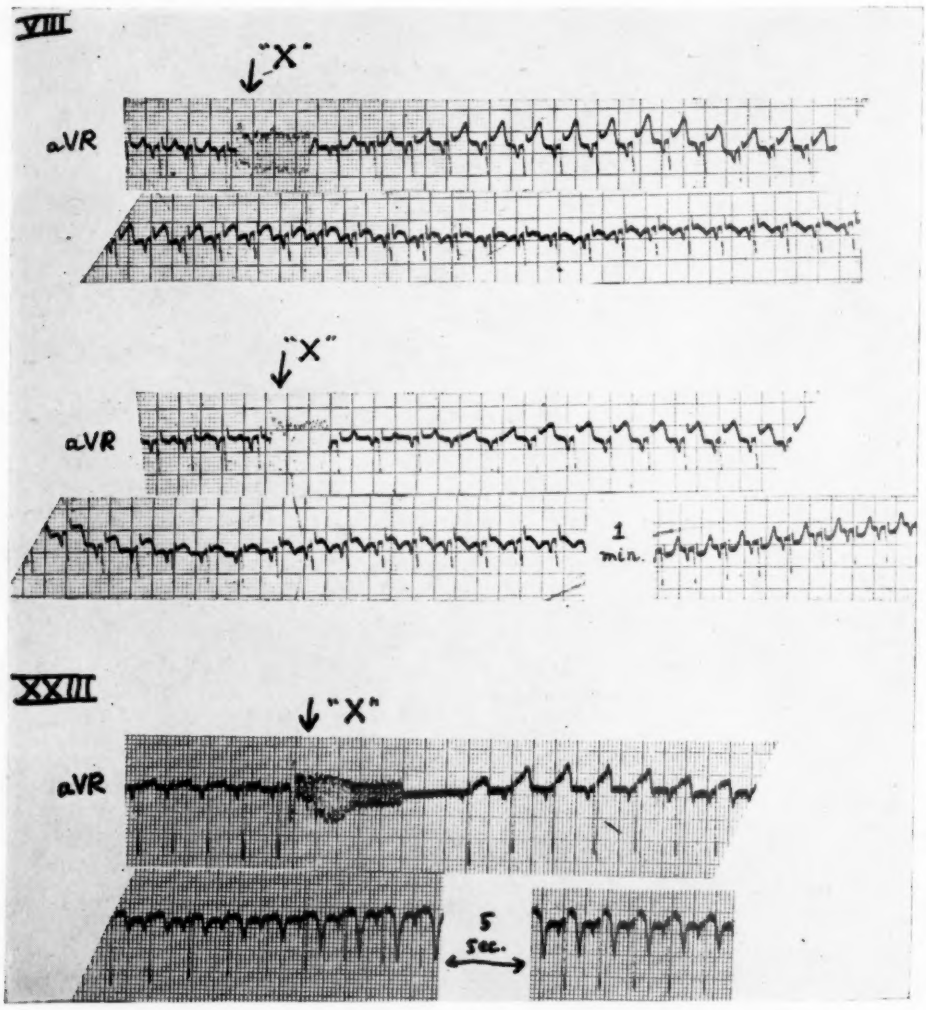

Fig. 3.—*Upper*, Effects of stimulating cardiovagal branches in Dog No. 8. Lead aVR. After 0.7 second stimulation, there is progressive elevation of ST and increase in size of T, followed by a return of ST to normal and inversion of T. The same result is obtained again after 0.55 second stimulation, returning to normal one minute later.

Lower, Lead aVR in Dog No. 23. After 1.0 second stimulation, there is cardiac standstill of 0.6 second (total asystole = 2 seconds), followed by ST elevation and T increase, and progressive T inversion.

in height and sharpening of the T waves where initially upright and displacement of the ST segments in a direction opposite to the T waves. In those leads where the T wave was initially inverted, the inversion became deeper and sharper and the ST segments became elevated. These changes strongly resemble those reported for acute coronary insufficiency in human beings (Fig. 4).

Stimulation of the stellate ganglion tended to abort the effects obtained by stimulation of the vagal branches. Pharmacologic studies with atropine and Adrenalin were made, but not in sufficient detail to be reported here.

These animal experiments indicate that stimulation of certain branches of the vagus had a profound effect upon the electrocardiogram producing arrhythmias and changes similar to those encountered in acute myocardial infarction in human beings. In some instances, death of the animal resulted because of

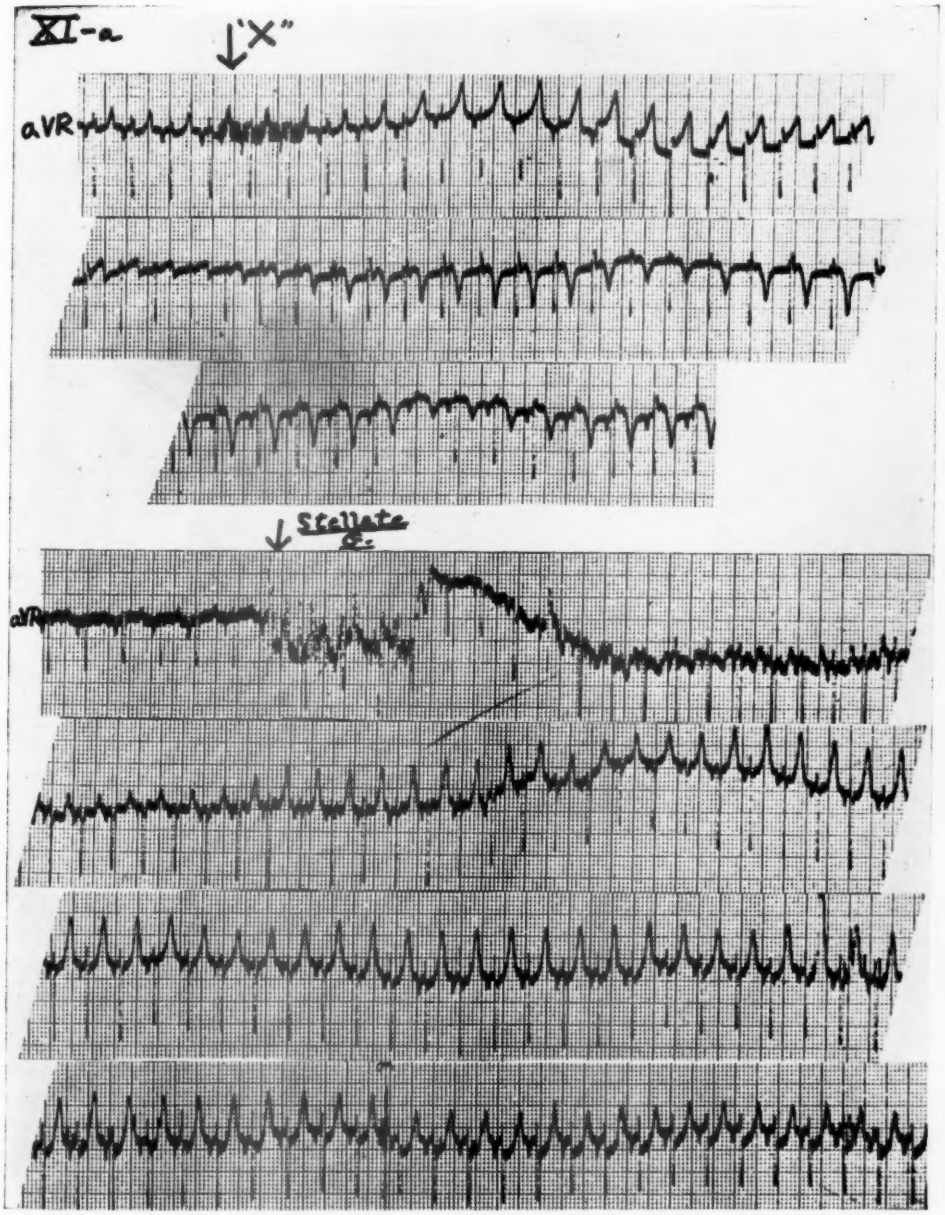

Fig. 4.—Comparison of cardiovagal and stellate stimulation. Lead aVR in Dog No. 11. At the arrow, the upper cardiovagal branches were stimulated for 0.8 second, followed by increase in height of T and ST elevations, then return of ST toward normal and T inversion. Stimulation of the intact stellate ganglion in this animal (at arrow) is followed by increase in height of T and ST depressions.

ventricular fibrillation preceded by these typical electrocardiographic changes (Fig. 5). Stimulation of the stellate ganglion produced electrocardiographic changes resembling acute coronary insufficiency.

Because neurogenic factors clinically seem to be important in precipitating angina, and because these experiments seem to support such a theory, we set out to apply these findings to human beings.

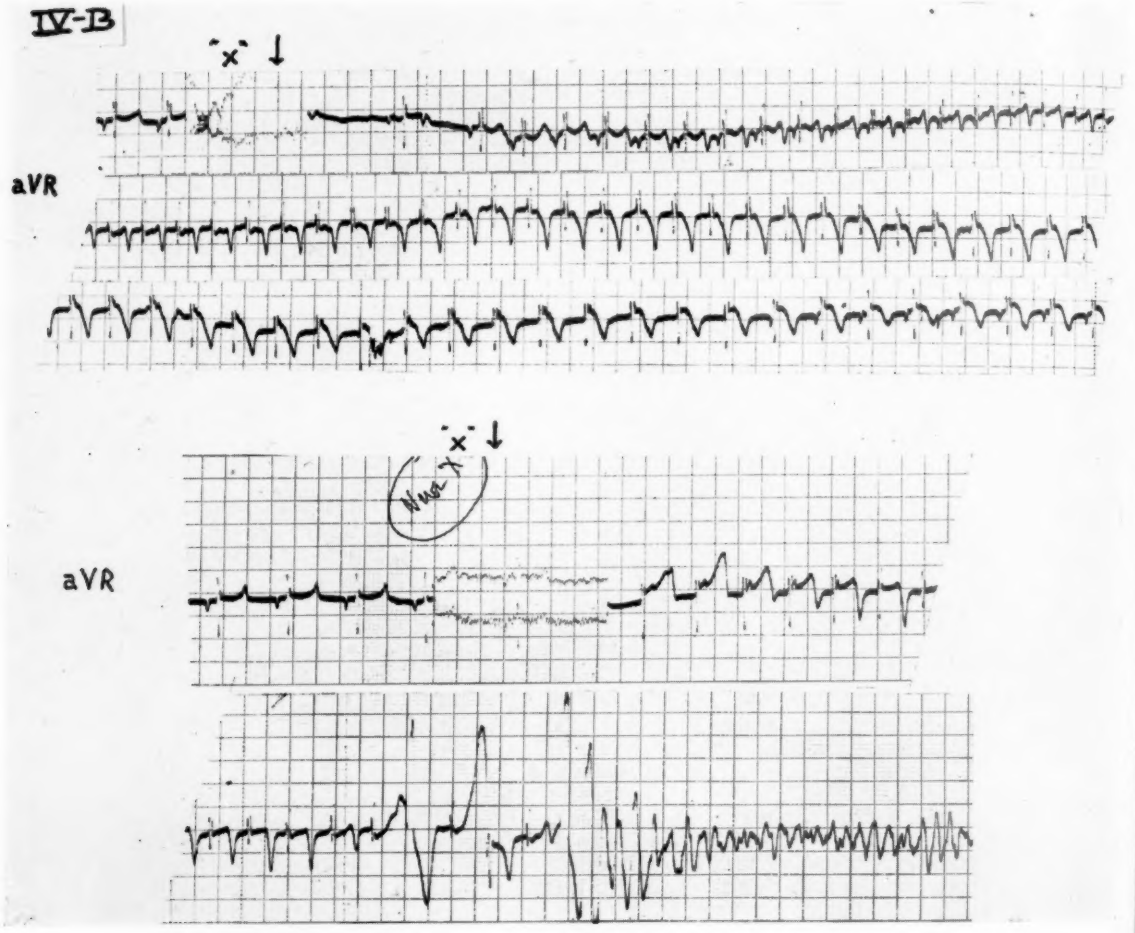

Fig. 5.—Effect of upper cardiovagal stimulation in Dog No. 4. Lead aVR. Stimulation at "*x*" for one second is followed by brief AV block, then an increase in heart rate. This is accompanied by deep symmetrical inversion of T and ST elevations.

When "*x*" is again stimulated, this time for 1.5 seconds, there is ST elevation, increase in height of T, T inversion, and fatal ventricular fibrillation.

To date, we have operated upon four patients. The first patient (*D*) had carcinoma of the left lower lobe of the lung and severe angina. With his consent, a left pneumonectomy was performed. In addition, the cardiac branches of the vagus nerve and the cardiac branches of the recurrent laryngeal nerve were severed (Fig. 6). The patient withstood the procedure well. Following operation, despite the fact that he now had one lung, he was able to engage in much more physical activity than he had before, without precipitating anginal discomfort. He died five months later as a result of metastasis of the carcinoma. During this time he was entirely free of anginal discomfort at rest and upon exertion. The second patient (*B*) had severe angina which did not respond to any type of medical treatment. He

was incapacitated and had been unable to work the preceding year. His activity was severely restricted. He used 25 to 30 nitroglycerin tablets daily. Following selective vagotomy (cardiac branches), he has returned to work and experiences no anginal discomfort. He is at present engaged in moderate physical activity. The chief problem is that of having him avoid strenuous activity which causes no symptoms but which we consider inadvisable for the present until there has been an opportunity for development of new collateral vessels.

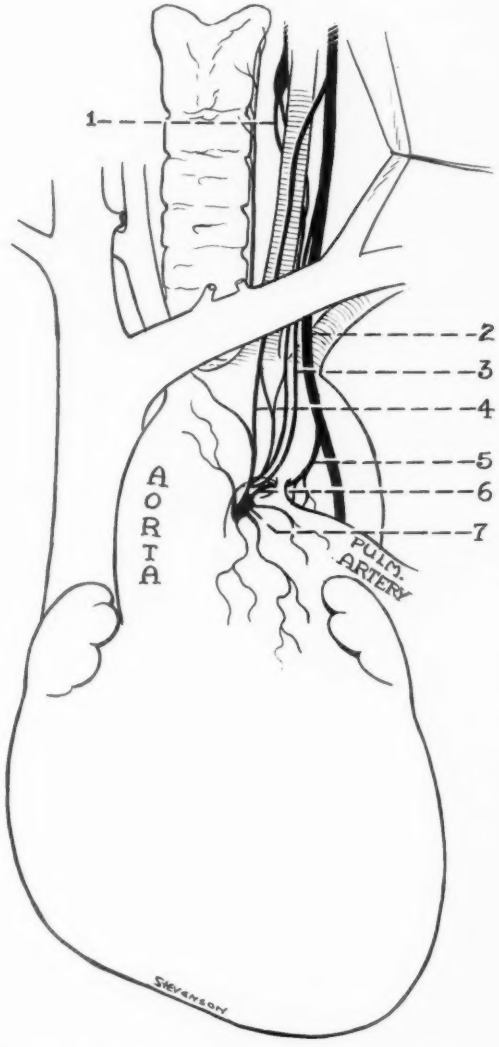

Fig. 6.—A composite drawing of several vagal dissections in human beings. *1*, Cervical sympathetic trunk; *2*, left main vagus nerve; *3*, and *4*, cardiac branches of left vagus passing to the ventricular plexus; *5* and *6*, recurrent laryngeal nerve with fibers to the cardiac plexus; *7*, cardiac or ventricular plexus.

The third patient (*C*), a retired railroad engineer, had severe angina pectoris precipitated not only by activity and exertion but by emotional stress as well. Following operation he gradually improved so that now he takes nitroglycerin on infrequent occasions and is able to engage in activity much greater than that tolerated for the six years previous to operation.

The fourth patient (*G*) was admitted with arresting angina pectoris which prevented his gainful occupation. Cardiovagal neurectomy completely relieved his anginal pain. Another discomfort which was eventually attributed to a small esophageal hiatal hernia was

relieved by phrenic crush. Two months after operation the patient engaged in the strenuous physical activity of digging a ditch 4 by 6 feet in size; this was followed two days later by acute myocardial infarction. He has made a satisfactory recovery from that episode.

All these patients were carefully selected from many who wished to undergo surgery. In all there was severe angina pectoris caused by definite coronary artery disease, as demonstrated by abnormal electrocardiograms either at rest or after exercise. Treatment with drugs, rest, and psychotherapy had been unsuccessful. An account of the responses of these patients to medical treatment is published elsewhere.[13] Greater details of history and electrocardiographic studies are available there.

It is too early to formulate final conclusions from these clinical results. We feel that the results are encouraging. They stress an important approach to the treatment of coronary disease for the following reasons: 1. Emphasis is placed upon surgical methods for improving the coronary circulation, rather than waiting for naturally developing collateral vessels. 2. Emphasis is placed upon the influence of neurogenic factors in the coronary circulation. Application of these methods to human beings has been applied with promising results.

Further work to determine the influence of selective nervous stimulation upon coronary blood flow, blood pressure, myocardial metabolism, and other factors is now being carried on. Eventually, perhaps, there may be a dual approach to the problem of improving coronary circulation consisting in the removal of pathways of constrictor impulses as the procedure here suggests and of anatomically increasing coronary blood supply by one of the surgical procedures now available or a variant thereof.

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CHANGES IN BLOOD CELLULAR ELEMENTS IN DOGS DURING HYPOTHERMIA

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HYPOTHERMIA stands out now as one of the quasiphysiologic methods by which body processes can be retarded to permit new surgical procedures. The surgical procedures considered for trial in the state of reduced body temperature are for the most part concerned with the heart and the aorta proximal to the renal arteries. This by no means outlines the extent of usefulness of hypothermia and, at the present time, such a general description of the possible applications of cooling would be impossible. It is enough to say that this experimental approach to selected problems in therapeutics offers promise enough to justify the intensive study currently directed to it.

We have not prepared a summary which traces the development of the present knowledge of the effects of subnormal body temperature. The authoritative contributions from Bigelow and his associates, Swan and associates, as well as many others, are readily available and need not be cited at this time. Although these earlier investigations in cooling have dealt with effects upon many organs and physiologic processes there remains an even greater number of problems which require study. One of these is the effect upon the blood cellular elements, and this constitutes the subject of the present report.

Our attention was first drawn to the possibility of changes in these components of the circulating blood when animals were observed to suffer hemorrhage into the intestinal tract after rewarming. This complication developed in over 50 per cent of the dogs which survived cooling to 27° C. and then were rewarmed. A second complication was observed in another group of dogs which had been cooled to the same temperature and then subjected to thoracotomy. There was a very high incidence of uncontrollable oozing from the incisions during the operative procedure. Those dogs that survived and were rewarmed frequently developed large hemothoraces and succumbed to this complication. It is surprising that a hemorrhagic diathesis which was such a prominent complication in this laboratory has not been observed by other investigators working in the field of hypothermia. While this question remains, it may be noted that several other authors[1, 2] have given casual mention to intestinal hemorrhage which developed in dogs that had been cooled. In addition to this, the postoperative bleeding into the pleural space which is known to have complicated the course of several surgical patients after cooling may be of special significance.[3]

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

METHOD

The mongrel dogs which were used were 8 months old or older and they had been in the laboratory colony less than one week. The only ones rejected were those with signs of some infection or malnutrition. In administration of the anesthetic there was some effort to avoid exciting the animal, and, as a rule, the solution of pentobarbital sodium was administered intravenously with little or no struggle. The average dose of this agent was 250 mg.; intubation of the trachea was carried out at once, and this was facilitated in some of the animals by the administration of small amounts of ether by open drop. A mixture of cyclopropane and oxygen was administered thereafter through a system which included a canister of soda lime for the absorption of carbon dioxide. The cyclopropane-oxygen mixture proved an ideal anesthetic in all experiments in which hypothermia was induced. Some difficulty was encountered in the control series, however, and it was necessary to administer small doses of diphenhydramine hydrochloride (Demerol) to maintain anesthesia at a satisfactory plane. All animals breathed actively throughout the course of the experiments and no device was employed for artificial ventilation.

The experiments were carried out with the dogs in the prone position. In addition to this they were tilted so that the extended hind legs were at a level 6 inches higher than the head. The dorsal surface of the thorax only was shaved and cracked ice was packed about all of the body except the head and neck. A slow circulation of brine through this loose ice pack speeded the cooling process.

The additional surgical procedures were of a minor nature. A short incision was made in the midline of the neck in order to expose the trachea. A tape ligature was passed around this structure and tied so that there was no escape of gas around the inlying tracheal catheter. A catheter was inserted into the superior vena cava in order that the venous pressure could be measured. All samples of venous blood drawn after the animal had been anesthetized were obtained through this system; the samples were collected in heparin tubes by gravity and syringes were not employed. A plastic cannula was inserted into the left femoral artery and this was attached to a mercury manometer. Changes in body temperature were measured by a mercury thermometer inserted into the sigmoid colon.

The samples of venous blood in heparin tubes were placed in a pan of cracked ice. The counting of erythrocytes, platelets,[4] and leukocytes,[5] preparation of blood smears, and determination of hematocrit[6] were completed within four hours after the samples were obtained.

RESULTS

The control experiments included all the details of the main group of experiments except the application of ice. All of the animals in both the control and the hypothermia groups were anesthetized, shaved, and cannulated in the same way, and the single variation in the standard protocol was the one noted in the description of the method pertaining to anesthesia. The dogs in the control series required somewhat more anesthetic agent and therefore the cyclopropane-oxygen mixture was supplemented with small amounts of diphenhydramine hydrochloride (Demerol) given intravenously. The dogs which were cooled were adequately anesthetized with cyclopropane and may have demonstrated in this way the narcotic effect of hypothermia. Respiratory acidosis of a severe degree resulted from the deep narcosis and the relatively low respiratory tidal volume. This unphysiologic state with the pH of the whole blood averaging 7.2 and the carbon dioxide tension 70 mm. Hg was present in nearly every animal in both groups.

Changes observed in the cellular elements of the blood of the control series are given in Table I.* The average changes are stated for each subdivision of the data, and in addition to this the range is given through which the changes were distributed. In the period of observation, which averaged 157 minutes, there was very little change in the hematocrit; the average was an increase of 0.5 volume per cent. The total counts of eosinophils rose in all but one of the

TABLE I. CHANGES OBSERVED IN CONTROL SERIES OF DOGS (5 EXPERIMENTS)

	DURATION OF EXP.	LEUKOCYTES	TOTAL EOSINOPHILS	PLATELETS	HEMATOCRIT	MCHC*	MCV†
Average	157 min.	Rise 132 per c.mm.	Rise 230.7	Fall 85,620 per c.mm.	Rise 0.5 vol. %	0	Rise 1.6 cu. microns
Range	140 to 175	−7,800 to +7,800	−22.2 to +444	−10,000 to −160,000	−10 to +11	−1.9 to +1.2	−2.5 to +5.4

*Mean corpuscular hemoglobin concentration.
†Mean corpuscular volume.

dogs, and the average of all of the changes was an increase of 230.7. The platelet count decreased in every control experiment with 85,620 being the average of this fall. The mean corpuscular hemoglobin concentration showed small changes in the individual experiments and their average was zero. The average of changes in the mean corpuscular volume was an increase of 1.6 cubic microns.

TABLE II. CHANGES OBSERVED IN DOGS COOLED BY IMMERSION (15 EXPERIMENTS)

	DURATION OF COOLING	MIN. TEMP.	LEUKOCYTES	TOTAL EOSINOPHILS	PLATELETS	HEMATOCRIT	MCHC	MCV
Average	100 min.	24.3° C.	Fall 8,656 per c.mm.	Fall 228	Fall 429,974 per c.mm.	Rise 9.0 vol. %	Fall 1.7 %	Rise 7.5 cu. microns
Range	46 to 187	25.3 to 22.5	−6,200 to −11,640	−122.1 to −421.8	−284,200 to −538,700	−4 to +25.5	−6.0 to +3.1	−9.2 to +20.5

The changes observed in the blood cellular elements of the 15 dogs subjected to cooling are listed in Table II. Again it will be noted that the average changes are given, and with this the range over which the changes were distributed. A reduction of body temperature to an average of 24.3° C. produced some marked effects, and these developed rapidly since the average duration of subnormal temperature was only 100 minutes. The hematocrit was increased in every dog but one, and the average of this rise was 9 volumes per cent.

*The following values are the averages obtained in this laboratory in dogs with normal body temperature, subjected to less than 15 minutes of the standardized anesthesia technique and minor surgical procedures of blood vessel cannulation:

Hematocrit	43.7 vol. per cent
Leukocytes	11,944 per c. mm.
Total eosinophils	275.8
Platelets	518,455 per c. mm.
Mean corpuscular hemoglobin concentration	32.2 per cent
Mean corpuscular volume	80.2 cu. microns.

The effect upon the total leukocyte count was a reduction in every experiment; the average fall was 8.656 per cubic millimeter. The total eosinophils, too, were decreased in every instance; the average reduction of these cells was 228. The examination of blood samples for platelets revealed the most outstanding effect of cooling in this species. The platelet count was greatly reduced in every dog; the average fall was 429,974 per cubic millimeter. Moreover, in two of the experiments no platelets were present in the samples of the circulating blood drawn when the maximum cooling effect had been obtained.

Blood samples from the hypothermic dogs showed that with cooling there was a fall in the mean corpuscular hemoglobin concentration. The reduction in this value averaged 1.7 per cent. The effect of cooling was to increase the mean corpuscular volume; the average of this change was 7.5 cubic microns.

SUMMARY

1. The standard methods of this laboratory for anesthetizing dogs and cooling them by immersion were employed in a group of 15 animals. A series of blood samples were drawn for observation of the effects of hypothermia on blood cellular elements. A control group of dogs with normal body temperature was subjected to the same type of anesthesia and minor surgical procedures and the same examinations of blood cellular elements were carried out.

2. Blood samples drawn from dogs in the control group showed slight change in the hematocrit and the total leukocyte count. The total eosinophil count rose and the platelet count fell somewhat in the control experiments.

3. Blood samples taken from the other animals showed that hypothermia produced the following changes: Both the hematocrit and the mean corpuscular volume rose. The total leukocyte count fell considerably and there was a fall in the total eosinophil count. A profound reduction occurred in the number of platelets, and in two of the experiments there were none of these elements in the circulating blood. The mean corpuscular hemoglobin concentration was also reduced.

4. A hemorrhagic diathesis has been noted in dogs cooled in this laboratory. Although the process of coagulation has not yet been studied directly, the results of these experiments suggest a relationship between the prolonged bleeding and the thrombocytopenia induced by hypothermia.

We wish to acknowledge the assistance given by Miss Jocelyn Simpson in preparation of the manuscript.

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VENTRICULAR FIBRILLATION IN THE HYPOTHERMIC STATE

V. General Discussion

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VENTRICULAR fibrillation constitutes the single greatest danger in the use of acutely induced hypothermia as an aid in cardiac surgery and, more particularly, as a means of permitting open surgery upon the bloodless heart. The incidence of this complication during brief periods of venous inflow occlusion and open operations through the right atrial wall or the main pulmonary trunk, both in animals and in man, has been reasonably low but, unfortunately, not absent. In contrast, experimental investigation of this techique for operations upon the opened ventricle has yielded the most discouraging results. In the studies of Montgomery and his group,[1] those of Radigan and his associates,[2] and those carried out in our laboratory,[3, 4] fibrillation has occurred almost uniformly. For this reason, those who are most enthusiastic about employment of hypothermia as an aid in cardiac surgery have been in agreement that it should not be used for intraventricular operations. One of the most important problems relating to hypothermia, therefore, concerns the prevention and treatment of ventricular fibrillation.

The Prevention of Ventricular Fibrillation.—Swan and his colleagues[5] have presented studies which indicate that hyperventilation with oxygen tends to reduce the likelihood of the occurrence of ventricular fibrillation, and most investigators are in agreement. In all of our own experimental studies and in the clinical use of hypothermia, hyperventilation with oxygen has been used. It is quite apparent, however, that hyperventilation will not protect hypothermic dogs from ventricular fibrillation when the right ventricle is opened during venous inflow occlusion or when certain other stimuli are given. Indeed, rough cardiac manipulation, ventriculotomy, and coronary sinus catheterization almost invariably produce fibrillation in spite of hyperventilation.[1-4] It has been necessary, therefore, to search for more effective methods.

Montgomery and his colleagues reported that the perfusion of the coronary arteries with acetylcholine or Prostigmin protected hypothermic dogs against fibrillation resulting from right ventriculotomy. We have had no experience with the use of acetylcholine in this fashion and only a small experience with coronary perfusion of dilute Prostigmin solution. Some of the animals subjected to coronary perfusion in the manner outlined by Montgomery and his

Aided by a contract between the Office of Naval Research, the United States Navy, and Indiana University, and by grants from the Indiana Heart Foundation and the James Whitcomb Riley Memorial Association.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

group did, however, develop ventricular fibrillation and we were struck with the difficulty of re-establishing adequate cardiac function even when fibrillation did not occur. The heart rate tended to be very slow and the contractions quite weak and we were not always successful even when cardiac massage and intracardiac administration of epinephrine were used.

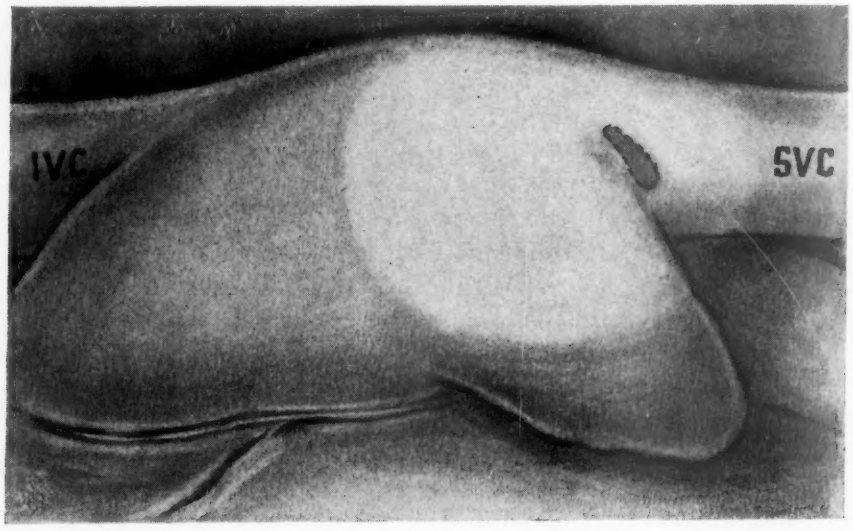

Fig. 1.—Drawing showing area infiltrated in atrial wall procaine block.

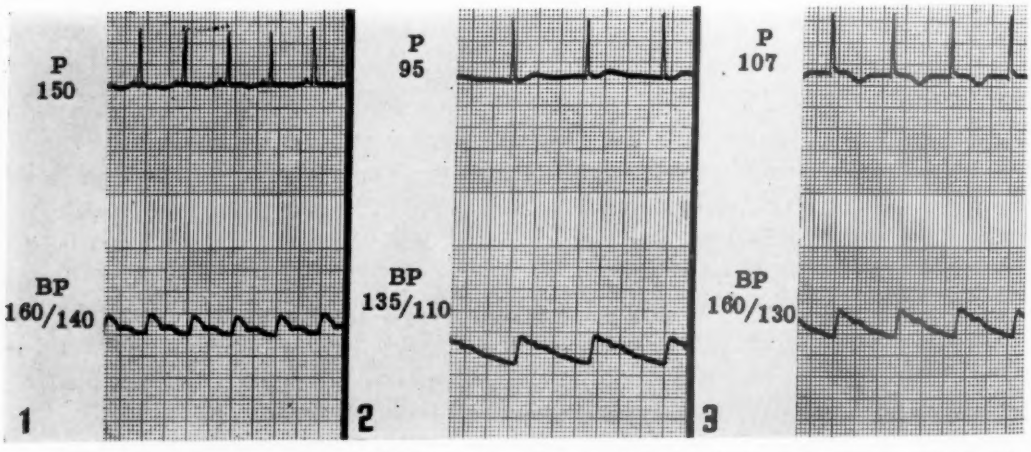

Fig. 2.—Effect of procaine injection of atrial wall upon electrocardiogram (Lead II), pulse, and blood pressure in normothermic dog No. 2. *1*, Before injection; *2*, after injection; *3*, twelve minutes later.

Sinoauricular Node Blockade.—When the right atrial wall is widely injected in and around the area of the sulcus terminalis in such a way as to infiltrate the atrial wall adjacent to the interatrial groove, the interatrial groove itself, the right lateral and anterior aspects of the superior vena cava at its point of union with the atrium, and the superior and right lateral aspects of the atrial appendage (Fig. 1), electrocardiographic evidence suggesting sinoauric-

ular node blockade is obtained. Normothermic dogs were anesthetized with 3 to 4 c.c. of 5 per cent thiopental sodium, intubated, and hyperventilated 35 to 45 times per minute with oxygen. The right chest was entered through the fourth intercostal space anteriorly and the pericardium was opened widely. The atrial wall was carefully infiltrated with 1 per cent procaine. Electrocardiographic and blood pressure tracings were made continually. The blood pressure was recorded with the Poly-viso cardiette and a strain gauge manometer connected with a plastic catheter introduced into the abdominal aorta through the femoral artery. In 70 per cent of the animals the P wave disappeared from the electrocardiographic tracing and in the remainder its voltage decreased considerably (Fig. 2). The pulse rate slowed strikingly, on an average of from

TABLE I. PULSE, BLOOD PRESSURE, AND ELECTROCARDIOGRAPHIC CHANGES IN NORMOTHERMIC DOGS WITH PROCAINE BLOCK OF RIGHT ATRIAL WALL

NO. OF DOG	PULSE RATE		BLOOD PRESSURE		ALTERATION IN P WAVE	DURATION P WAVE CHANGES IN MINUTES	PULSE AND BLOOD PRESSURE AT TIME P WAVE REVERTED TO PREINJECTION CONFIGURATION	
	BEFORE	AFTER	BEFORE	AFTER				
1	145	98	165/125	125/85	Lower voltage	9	120	145/100
2	150	95	160/140	135/110	Disappeared	12	107	160/130
3	150	93	160/135	155/120	Disappeared	11	90	165/135
4	165	115	170/110	140/190	Lower voltage	8	140	135/95
5	130	55	180/135	110/78	Lower voltage	16	65	170/110
8	150	89	195/135	155/110	Disappeared	9	94	190/127
9	135	80	195/135	142/90	Disappeared	6	125	195/140
10	136	75	175/110	155/110	Disappeared	9	95	135/85
11	136	100	185/95	175/75	Disappeared	6	106	180/95
13	136	115	155/105	180/116	Disappeared	11	125	145/80

143 to 91 per minute (Table I). The blood pressure similarly fell, but only moderately. The average preinjection blood pressure was 174/122 and the average pressure after injection 165/98. The duration of the altered electrocardiographic tracing was brief, ranging between six and twelve minutes, and by the time the P wave had returned to its preinjection configuration, the arterial blood pressure had also increased to almost the initial level. The pulse rate

TABLE II. PULSE, BLOOD PRESSURE, AND ELECTROCARDIOGRAPHIC CHANGES IN HYPOTHERMIC DOGS WITH PROCAINE BLOCK OF RIGHT ATRIAL WALL

NO. OF DOG	TEMPERATURE OF DOG IN ° C.	PULSE RATE		BLOOD PRESSURE		ALTERATION IN P WAVE	DURATION P WAVE CHANGES IN MINUTES	PULSE AND BLOOD PRESSURE AT TIMES P WAVE REVERTED TO PREINJECTION CONFIGURATION	
		BEFORE	AFTER	BEFORE	AFTER				
6	28.5	86	65	143/110	150/100	Disappeared	12	75	150/115
7	27	55	33	130/95	85/50	Lower voltage	15	33	105/65
12	25.5	43	33	167/126	140/98	Disappeared	20	45	140/110
14	28	75	56	160/125	145/100	Lower voltage	13	60	135/95
15	27	65	52	165/115	145/90	Disappeared	24	58	155/100
16	27	75	51	134/100	135/65	Disappeared	27	57	125/85
17	26	160	45	140/105	135/80	Lower voltage	16	54	135/95
18	25.5	62	44	150/120	105/68	Disappeared	20	45	125/85
19	24	63	34	125/95	100/65	Disappeared	16	24	110/80
20	28	86	46	143/110	130/70	Disappeared	17	75	160/135

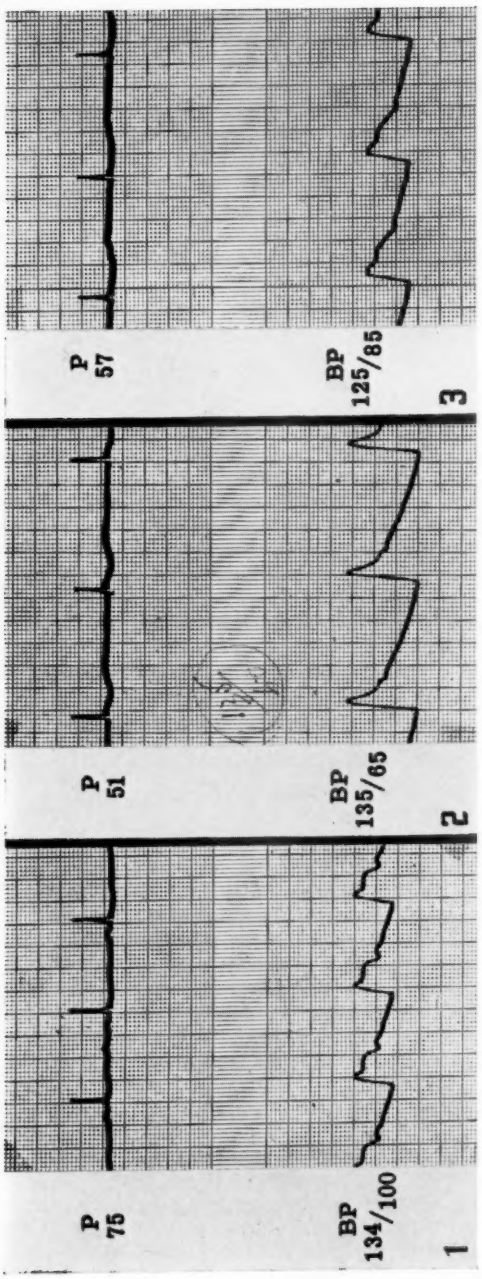

Fig. 3.—Effect of procaine injection of atrial wall upon electrocardiogram (Lead II), pulse, and blood pressure in hypothermic dog No. 16. Rectal temperature 27° C. *1*, Before injection; *2*, after injection; *3*, twenty-seven minutes later.

remained somewhat slower than the preinjection rate for some time longer. During the period of sinoauricular node blockade one gained the impression, from tapping the ventricle of the heart and carrying out other manipulations, that cardiac irritability was definitely reduced below normal.

A similar but more prolonged effect is noted when hypothermic dogs are injected in the same fashion. A group of animals in which hypothermia was induced were studied in precisely the same manner as those just mentioned. After induction of anesthesia, intubation, and institution of hyperventilation with oxygen, each dog was cooled to a body temperature of 30° C. by immersion in a bath of cracked ice and water. By the time the heart was exposed and the atrial wall infiltrated, the temperature had fallen to from 24° to 28.5° C. Upon accomplishment of a good injection, the P wave disappeared (70 per cent) or underwent a decrease in voltage (30 per cent) (Fig. 3). The pulse rate decreased considerably, on an average of from 67 to 46 per minute. The blood pressure decreased moderately. The average blood pressure before injection was 146/110 and after injection was 125/78. The duration of effect, as measured by altered P waves, was somewhat longer than in normothermic dogs, varying from 12 to 27 minutes. As in the normothermic group, the blood pressure by this time had risen, but not to the preinjection level. Some relative slowing of the pulse tended to persist for a longer period of time.

TABLE III. EFFECT OF PROCAINE BLOCK OF SINOAURICULAR NODE UPON INCIDENCE OF VENTRICULAR FIBRILLATION IN HYPOTHERMIC DOGS STIMULATED BY CARDIAC MANIPULATION*

NO. OF DOGS	TEMPERATURE OF DOGS IN ° C.	AVERAGE PERIOD OF CARDIAC VENOUS INFLOW OCCLUSION IN MINUTES	PER CENT FIBRILLATION
	Untreated Controls		
6	24.5 to 27	4.8	100
	Controls (Saline Injection of Atrial Wall)		
2	26.8 to 28	6.5	100
	(Treated Procaine Injection of Atrial Wall)		
8	23.5 to 27.5	10.4	0

*3 Controls and 3 treated had rough external manipulation; 3 untreated controls, 2 saline-injected controls and 5 treated dogs had finger manipulation of ventricular septum through right atrium and tricuspid valve.

In dogs cooled moderately, such procaine injection of the atrial wall has proved remarkably effective in preventing ventricular fibrillation.[3] When such animals were subjected during venous inflow occlusion to rough external manipulation of the heart or to vigorous massage of the ventricular septum by means of a finger passed through the right atrium and tricuspid valve, all the control animals, and none of those treated by procaine injection, underwent fibrillation (Table III). When animals were similarly studied with the use of right ventriculotomy and placement of sutures in the ventricular septum and venous inflow occlusion of ten minutes or more, similar results were obtained. All the untreated controls and two-thirds of the controls given a saline injection of the atrial wall developed ventricular fibrillation, while fibrillation did not occur in any of those treated by procaine injection (Table IV). Experiments at lower

TABLE IV. EFFECT OF PROCAINE BLOCK OF SINOAURICULAR NODE UPON INCIDENCE OF VENTRICULAR FIBRILLATION IN HYPOTHERMIC DOGS STIMULATED BY RIGHT VENTRICULOTOMY AND PLACEMENT OF SUTURE IN VENTRICULAR SEPTUM

NO. OF DOGS	TEMPERATURE OF DOGS IN ° C.	AVERAGE PERIOD OF CARDIAC VENOUS INFLOW OCCLUSION IN MINUTES	PER CENT FIBRILLATION
	Untreated Controls		
6	27 to 28	4.2	100
	Controls (Saline Injection of Atrial Wall)		
6	25.5 to 28	6.8	67
	Treated (Procaine Injection of Atrial Wall)		
11	24.6 to 28.3	10.4	0

body temperatures, ranging from 19 to 22.5° C., have revealed similar protection.[4] Over half of the untreated controls and only 1 out of 16 with procaine infiltration developed fibrillation (Table V).

This type of experiment has been repeated by Radigan, Lombardo, and Morrow.[2] In dogs cooled to from 26° to 28° C. and subjected to vena caval occlusion for eight minutes, right ventriculotomy and placement of sutures in the ventricular septum, ventricular fibrillation occurred in 18 of 20 control animals, and, in spite of the fact that all were resuscitated, the ultimate mortality was 65 per cent. In contrast, none of the 20 animals which were protected by procaine injection of the superior atrial-caval junction developed fibrillation and all but 1 animal survived the experiment. All their animals given intravenous procaine or intrapericardial procaine underwent ventricular fibrillation and died.

TABLE V. EFFECT OF PROCAINE BLOCK OF SINOAURICULAR NODE UPON INCIDENCE OF VENTRICULAR FIBRILLATION IN DOGS WITH LOW BODY TEMPERATURES SUBJECTED TO RIGHT VENTRICULOTOMY AND PLACEMENT OF SUTURE IN VENTRICULAR SEPTUM

NO. OF DOGS	TEMPERATURE OF DOGS IN °C.	AVERAGE PERIOD OF CARDIAC VENOUS INFLOW OCCLUSION IN MINUTES	PER CENT FIBRILLATION
	Untreated Controls		
16	19.5 to 22.5	8.2	56
	Treated (Procaine Injection of Atrial Wall)		
16	19 to 22.5	10.3	6

Similar protection has been observed by Lian and his associates[6] who, utilizing this maneuver, were able to perform ventriculotomy in dogs with drug-induced "artificial hibernation" without ventricular fibrillation.

Treatment of Ventricular Fibrillation.—There has been general agreement that the treatment of ventricular fibrillation occurring in the hypothermic state is more difficult than its treatment in normothermic circumsances. Swan and his colleagues[5] were unsuccessful in stopping such fibrillation with the use of electric shock and presented studies showing that perfusion of the coronary arteries with a weak solution of potassium chloride was effective. In our own hands, such use of potassium chloride does convert fibrillation to a standstill but conversion of the standstill to a normal rhythm often has not been achieved.

The use of cardiac massage or calcium chloride under such circumstances not infrequently leads to recurrence of fibrillation. Whether or not these observations are significant, we do not know, but a small group of dogs treated by procaine infiltration of the atrial wall and potassium chloride infusion of the coronaries had successful conversion of fibrillation to a standstill and, with cardiac massage, to a normal rhythm.

We have recently carried out some experiments[7] dealing with treatment of ventricular fibrillation induced in hypothermic animals by coronary air embolism. In these experiments advantage was taken of a number of recent contributions. One was the demonstration by Geoghegan and Lam[8] that air in the coronary arteries can in a large measure be driven from them by cardiac message. Another was the observation of Kirby and his colleagues[9] that higher voltages may be successful in bringing about conversion when shock with the 110 volts current commonly used in the past has been unsuccessful. Kirby, Jensen, and Johnson[10] were able to defibrillate the hearts of hypothermic dogs using 220 volts. A third is the interesting observation,[7] also made by Lewis,[11] that conversion is more likely when weak fibrillation is strengthened by the intracardiac injection of epinephrine. In our experiments dogs were cooled to from 25° to 26.5° C. and, after the venae cavae and the ascending aorta were clamped, air was injected into the left ventricle. Gross air embolism and ventricular fibrillation followed. Some animals were subjected to right ventriculotomy and placement of sutures in the ventricular septum. The hyperventilation with oxygen was continued and the heart was massaged vigorously after the vena caval clamps were released. In many, the air was completely driven from the coronary arteries. When air remained in one or more branches of the coronary arteries, it was possible to milk it out after cutting across the terminal portion of the blocked artery. When fibrillation was weak, it was strengthened by the injection into the right ventricle of dilute epinephrine solution (a few cubic centimeters of 1:20,000). Successful restoration of normal rhythm was accomplished in all animals when three successive electric shocks were administered using 170 volts and a duration of one-tenth second.

Occasionally, the injection into the right ventricle of a dilute solution of epinephrine may be useful in restoring strong cardiac contraction after successful conversion from ventricular fibrillation.

In these experiments procaine blockade of the atrial wall seemed to have some beneficial effect (Tables VI and VII). When the injection was carried out before the expected onset of fibrillation, 1 of 4 animals did not develop fibrillation. Fibrillation occurred in all the other animals. No recurrence of fibrillation developed after its successful conversion in any animal treated by injection either before or after onset of fibrillation. In contrast, fibrillation did recur in 5 of the 14 animals with no atrial wall injection (Tables VI and VII). In 1 animal, fibrillation recurred 9 times but not after the tenth conversion which followed sinoauricular node blockade.

Extrinsic Cardiac Innervation and Ventricular Fibrillation.—We have carried out some investigations relating to the possible influence of extrinsic cardiac

innervation upon ventricular fibrillation in the hypothermic state, since we could not be sure whether the protective value of right atrial procaine block resulted from sinoauricular node paralysis alone, blockade of the extrinsic nerves which enter the heart almost totally through the area injected, or a combination of both.[12] In these studies, dogs were cooled to approximately 26° to 27° C. and subjected to venous inflow occlusion for 10 minutes, right ventriculotomy,

TABLE VI. EXPERIENCES WITH TREATMENT OF CORONARY AIR EMBOLISM AND VENTRICULAR FIBRILLATION IN THE HYPOTHERMIC DOG BY CARDIAC MASSAGE, INTRACARDIAC EPINEPHRINE, AND ELECTRIC SHOCK

NO. OF DOGS	TEMP. OF DOGS IN °C.	AVERAGE VOLUME AIR INJECTED (IN C.C.)	AVERAGE INTERVAL BETWEEN INJECTION OF AIR AND ONSET OF FIBRILLATION IN MINUTES	AVERAGE DURATION OF FIBRILLATION IN MINUTES BEFORE INSTITUTION OF MASSAGE	AVERAGE PERIOD OF MASSAGE IN MINUTES NECESSARY FOR CLEARING CORONARIES OF AIR	AVERAGE DURATION OF FIBRILLATION IN MINUTES	REMARKS
			Sinoauricular Node Injection Prior to Expected Onset of Fibrillation				
4	26-26.5	4.9	4.8	2.5	2.1	9	One did not fibrillate; others resuscitated
			Sinoauricular Node Injection After Onset of Fibrillation				
6	26-26.5	3.9	2	3.3	5.3	9.8	Fibrillation in all; all resuscitated
			*No Sinoauricular Node Injection**				
10	25-26.5	4.2	0.8	4.75	2.3	13	Fibrillation in all; one had fibrillation twice; two 4 times; all resuscitated

*One had injection of the sinoauricular node after fibrillation recurred 4 times.

TABLE VII. EXPERIENCE WITH TREATMENT OF CORONARY AIR EMBOLISM AND VENTRICULAR FIBRILLATION IN HYPOTHERMIC DOGS SUBJECTED TO RIGHT VENTRICULOTOMY AND PLACEMENT OF SUTURES IN VENTRICULAR SEPTUM BY CARDIAC MASSAGE, INTRACARDIAC EPINEPHRINE, AND ELECTRIC SHOCK

NO. OF DOGS	TEMP. OF DOGS IN °C.	AVERAGE VOLUME AIR INJECTED IN °C.	AVERAGE INTERVAL BETWEEN INJECTION OF AIR AND ONSET OF FIBRILLATION IN MINUTES	AVERAGE DURATION OF FIBRILLATION IN MINUTES BEFORE INSTITUTION OF MASSAGE	AVERAGE PERIOD OF MASSAGE IN MINUTES NECESSARY FOR CLEARING CORONARIES OF AIR	AVERAGE DURATION OF FIBRILLATION IN MINUTES	REMARKS
			Sinoauricular Node Injection After Onset of Fibrillation				
4	26-27	4.1	2.2	5.6	2.2	7.5	Fibrillation in all; no recurrence of fibrillation; all resuscitated
			No Sinoauricular Node Injection				
4	25.5-27	4.4	1.1	5.2	2	27	Fibrillation in all; fibrillation recurred 3 times in one, 10 times in another*; all resuscitated

*Before the last successful defibrillation sinoauricular node was injected.

and placement of sutures in the ventricular septum (Table VIII). When the sympathetic nerve supply of the heart was completely extirpated, no fibrillation occurred. Peculiarly enough, following excision of the upper three thoracic and stellate ganglia, no consistent change in pulse rate was noted. Slowing of the pulse does occur in normothermic dogs after sympathetic denervation. A slight drop in blood pressure was noted in 3 of 5 animals studied. When the ganglion-blocking agent, Arfonad, was given, the incidence of ventricular fibrillation was 13 per cent. No animal developed fibrillation during ventriculotomy or venous inflow occlusion. Two underwent fibrillation during massage instituted later for weak cardiac contractions. The Arfonad brought about a slight decrease in pulse rate, from 70 to 65, and the average blood pressure fell from 153/111 to 60/26. When bilateral vagotomy was accomplished by

TABLE VIII. EFFECT OF EXTRINSIC CARDIAC INNERVATION UPON INCIDENCE OF VENTRICULAR FIBRILLATION IN DOGS COOLED TO APPROXIMATELY 26° TO 27° C. AND SUBJECTED TO VENOUS INFLOW OCCLUSION FOR 10 MINUTES, RIGHT VENTRICULOTOMY, AND PLACEMENT OF SUTURE IN VENTRICULAR SEPTUM

TYPE OF ALTERATION OF NERVE SUPPLY	NO. OF DOGS	PER CENT DEVELOPING FIBRILLATION
Bilateral upper dorsal sympathetic and stellate ganglionectomy	15	0
Injection of intra-atrial or intravenous Arfonad	15	13
Bilateral section of cervical vagosympathetic trunks	15	80
Stimulation of the right vagus	15	33 (entire group) 17 (excluding 3 in which vagal escape occurred)
Bilateral division of vagi distal to caudate ganglia	15	20
Bilateral upper dorsal sympathetic and stellate ganglionectomy and bilateral division of cervical vagosympathetic trunks	15	40

sectioning both cervical vagosympathetic trunks, a procedure which divides the vagus but does not, as far as we know, interfere with the sympathetic nerves to the heart, 80 per cent of the animals developed ventricular fibrillation. This procedure brought about no consistent change in pulse rate and a modest elevation of blood pressure in 3 of 5 animals. Stimulation of the right vagus nerve was associated with marked slowing of the pulse rate and some drop in blood pressure. When a persistent bradycardia attended stimulation, fibrillation appeared in only 2 of 12 animals. Fibrillation occurred in an additional 3 in which vagal escape was noted. Our studies of vagal stimulation thus confirm the beneficial effect of this maneuver noted by Montgomery and his associates.[1] In one group, the vagi were divided distal to the caudate ganglion. The level of division was below the cardiac depressor nerve and it is difficult to understand why the incidence of fibrillation in this group was only 20 per cent. The hypothesis that such vagal section initiates an axon reflex with cardiac vagal stimulation was not borne out by pulse and pressure studies. The blood pressure did not fall and there was no persistent bradycardia response. In some animals a total cardiac extrinsic denervation was performed. Forty per cent of them fibrillated.

These observations clearly demonstrate that sympathetic denervation, paralysis with the sympathetic ganglion-blocking agent, Arfonad, and vagal stimulation, are all of real benefit in reducing the incidence of ventricular fibrillation. The vagally denervated heart undergoes fibrillation frequently, the totally denervated heart less frequently.

Utilizing the knowledge gained concerning the prevention and treatment of ventricular fibrillation in the hypothermic state, we have been able to make and close ventricular septal defects during venous inflow occlusion and hypothermia without mortality.[13]

SUMMARY

Recent observations have made it clear that certain maneuvers bring about remarkable protection against ventricular fibrillation in the hypothermic state. Of these, it would appear that procaine infiltration of the right atrial wall and intravascular administration of Arfonad would have particular clinical applicability. A small experience suggests that procaine infiltration of the atrium has a similar effect in man to that noted in dogs. As yet we have made no observation with Arfonad in hypothermic patients. Experimentally produced ventricular fibrillation can be dealt with successfully under hypothermic conditions by massage, adequate ventillation, conversion of weak to strong fibrillation with epinephrine, relatively high voltage shock, and atrial wall blockade to minimize the chance of recurrence. Coronary air embolism in hypothermic dogs and the ventricular fibrillation which follows can be managed with success by these same measures, plus milking out through the cut terminal branch of the blocked artery of any air remaining after vigorous massage.

Stimulation and denervation experiments indicate that sympathetic block and vagal stimulation reduce the incidence of ventricular fibrillation when hypothermic dogs are subjected to venous inflow occlusion and right ventriculotomy.

Procaine infiltration of the right atrial wall produces in normothermic dogs P wave, pulse and blood pressure changes similar to those noted in hypothermic animals after atrial wall injection.

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DIRECT-VISION SUTURE OF INTERATRIAL SEPTAL DEFECT DURING HYPOTHERMIA

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I. INTRODUCTION

THE story of the successful surgical conquest of interauricular septal defect is a satisfying one in many ways since it exemplifies important principles in modern medical progress. In the first place, many individuals contributed either by elaborating basic concepts or devising new techniques, each adding in turn to the growing potential of the surgical repair of the lesion. Second, means were devised for the creation of an experimental lesion and thus almost all the new ideas could be and were extensively tested in the laboratory before application was attempted in the human being. Third, progress was rapid because, in modern surgery, free exchange of ideas and information is facilitated by frequent meetings; there is no "classified" data, no "iron curtain" to hide the work of one from the others. Fourth, the problem was not isolated, but was a facet of a bigger problem, heart surgery in general. As knowledge and experience in general principles in the larger field increased, new approaches to the solution of the specific problem became possible. Finally, as one might expect, once basic methods were elaborated, more than one good technique emerged. Time and experience must eventually decide if one or another is best.

The development of a technique for the creation of an experimental lesion simulating the human defect was a primary step of importance. Martin and Essex,[1] in 1948, attempted incision of the septum across the right auricle, but most of the defects healed spontaneously. The same fate met small excisions using a noncrushing clamp devised by Dodrill.[2] The posterior approach of Blalock and Hanlon[3] created a persistent defect, but one which lay anatomically posterior and superior to the common site of the clinical lesion. At about this time, 1949, having met similar lack of success in our laboratory with incisions, punches, and small, blind excisions, we turned to an open method, excising under direct vision most of the septum via the empty right auricle during temporary occlusion of the blood flow entering the heart.[4] This technique of inflow occlusion had been known for forty years. Haecker[5] (1907), Laewen and Sievers[6] (1910), Carrel[7] (1910), and Jeger[8] (1913) had all investigated the method experimentally. Large defects which remained patent indefinitely could be created in this fashion with a low operative mortality rate. Circulatory occlusion was limited to one and one-half minutes, and air embolism to the

Experimental aspects aided in part by a grant from the United States Public Health Service (H-1559), and in part by a grant from The American Heart Association.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, June 5, 1955.

coronaries was eliminated by flooding the heart with Ringer's solution to empty the heart of air before the auricular incision was closed. This method has subsequently been widely used for experimental evaluation of closure techniques.[9-13] With the addition of hypothermia, inflow occlusion, as used to create these experimental defects, was later developed as a method for closure under direct vision (vide infra).

Other methods for creation of a lasting defect were later developed.[15, 16] Of particular importance was the technique described by Gross and associates,[17] in 1953, since their development of the use of an atrial well resulted in a method which, like inflow occlusion, was subsequently utilized as a closure technique.

The first experimental attempt at repair of an atrial septal defect was that of Cohn in 1947[18]; his idea was the use of a patch of auricle wall. Intrigued by this technique, but disturbed because it exposed to the right auricular lumen the cut auricular muscle which we thought might lead to serious thrombosis, we developed in our laboratory the concept of utilizing auricle wall, but inverting it rather than detaching it, so that only endothelialized surfaces were exposed to the blood stream.[4] The auricular tips were used in these experiments. The technique was only partially successful, but a principle was established which later proved of importance. This was the concept of the "doughnut" lumen of the auricles, which, it was shown, did not interfere with the flow of blood through the chambers. This principle was subsequently utilized by Bailey and co-workers,[19] in 1952, who modified the technique to involve suture of the lateral wall of the auricle to the rim of the defect guided by a finger inside the auricle, and by Shumacker and associates,[20] who added to the procedure first a pericardial and later a plastic sac attached to auricle wall.

Meanwhile, another variation of the patch idea of Cohn was being explored. Instead of a plug of heart muscle, inert plastic buttons were examined to see if they would not excite thrombosis. Hufnagel and Gillespie,[15] in 1951, described an ingenious method for the application of two nylon buttons. Swan,[12] in 1953, described a modification of his original button technique which left a polyethylene sheet over the defect inside the right auricle, and described the remarkable process which first covered the plastic with fibrin, and then by endothelialization converted the enclosure to a smooth, glistening membrane. Later, Donald and associates[21] used polyvinyl sponge, while Miller and co-workers,[22] Pomeranz, Watkins, and Gross,[23] and Bahnson and Baker[24] evaluated a variety of tissues and plastic materials. These experiments demonstrated the important fact that whereas pericardial grafts or other autogenous or homologous free-tissue grafts excite thrombosis and are replaced by fibrous scars, a variety of plastic materials are well tolerated in the lumen of the auricle and excite a process which results in their becoming completely encased in a smooth endothelialized membrane. If a proper technique to position such a patch is utilized, complete closure of the defect is possible. Utilizing this principle, Kirklin and co-workers[25] reported a very successful clinical series in 1955 placing the polyvinyl sponge by means of the Gross well.

Another technique for closure which suggested itself was direct suture. This might be performed from within or from without. The latter method had been suggested by Murray[26] in 1948, but his results were unconvincing. Interestingly, the more difficult internal suture was accomplished first. In 1953, Gross and associates,[17] using the well, and Lewis and Taufic[10] using direct vision during hypothermia, sutured previously created experimental defects. Gross applied this method to a patient in April and Lewis in September of the same year. Since that time, Muller and co-workers[27] have reported excellent success with the suture method via the well, Lewis and associates[28] have enlarged their experience, and we[29, 30] have also reported on the experimental and clinical application of the direct-vision suture method. It has become apparent that very large human atrial septal defects can be closed with success by direct suture without need for plastic prostheses in most instances.

The last successful method to be evolved seems, in retrospect, that it should have been the first. This is the circular suture of Söndergaard,[32] subsequently given clinical trial by Björk.[31] A large suture is passed through the upper edge of the ventricular septum or the lower portion of the atrial septum, and then tied down in a previously developed plane between the right pulmonary veins and the posterior aspect of the right atrium, thus squeezing closed the entire atrial septum.

As a result of all these interrelated endeavors, it would appear, at the present time, that there are at least five clinically applicable techniques which give satisfactory results at acceptable risk rates. "Atrio-septo-pexy" (Bailey), direct suture using the well of Gross and Muller, polyvinyl sponge applied via the well (Kirklin), and circular suture (Söndergaard and Björk) are all blind or closed heart methods in which the manipulation is carried out by feel. The proved open method is that of suture under direct vision in the open heart during hypothermia (Lewis and Swan). The use of the extracorporeal circulation as another direct-vision technique awaits further evaluation.

It is our opinion that direct vision is an important fundamental principle of surgery. It is for that reason that we have concentrated our effort in the exploration of one modality, hypothermia, which allows a limited time of intracardiac vision, as a technique for the clinical solution to this particular problem. It was our thought that if repair of this lesion could be accomplished under direct vision at a mortality rate comparable with indirect methods, it would be the preferable technique. This report submits the evidence for our opinion that this has been accomplished.

II. DIAGNOSIS AND INDICATIONS FOR OPERATION

The clinical features of patients with atrial septal defect have been elaborated more precisely in recent years so that now the clinical diagnosis may be established with a high degree of accuracy. Typically, the patient presents as an acyanotic child or adult without definite symptoms or with slight to moderate shortness of breath and fatigue with exertion. Examination usually reveals a slight individual, usually without deformity of the precordium. There is, however, evidence of a hyperdynamic cardiac impulse and of cardiac enlarge-

ment. Usually, no thrills or shocks are transmitted to the palpating hand. Auscultation reveals a rough systolic murmur of grade II or III intensity on a grade VI basis, of maximum intensity in the second left intercostal space and well transmitted toward the shoulders. The second heart sound in the second left intercostal space is widely reduplicated. There is also not infrequently a diastolic murmur audible over the tricuspid area, which, on rare occasions, has a presystolic component. This combination of findings in a child or young adult is highly suggestive of a diagnosis of an atrial septal defect.

The electrocardiogram is of great aid in further establishing this diagnosis. Partial right-bundle branch block is present in 90 to 95 per cent of this type of patient, and its absence would throw doubt on the diagnosis.

Fluoroscopic evaluation is also of great importance. The lung fields reveal a plethora that is noted well out toward the periphery, and the main right and left pulmonary arteries are dilated. The amplitude of the pulsations over these vessels is often increased but this is usually not marked. The aorta is inconspicuous. The heart is moderately to considerably enlarged with evidence of great enlargement of the right atrium and right ventricle. There is no clear-cut evidence of enlargement of the left atrium or left ventricle.

The patient revealing these findings will have an atrial septal defect of the secundum type in a very high percentage of cases. In the child below 2 years of age and in the patient over 45, however, the pattern may not be as clear-cut as this description would suggest, and the clinical diagnosis must be confirmed with cardiac catheterization.

The differential diagnosis between atrial septal defect and anomalies of pulmonary venous connections either as a sole defect or in association with an atrial defect continues to remain most difficult, if not, at times, impossible, although utilization of dye dilution curves is reported to be helpful. Catheterization alone will not differentiate these lesions.

There remains one other point of importance in the preoperative diagnosis, namely, identification of the type of defect frequently referred to as the persistent ostium primum.

Fundamentally, the embryologic classification described by Patten[33] divides atrial defects into two groups, those caused by failure of development of the septum primum and those caused by arrest of the septum secundum. These two different embryologic faults result in quite separate anatomic lesions and completely different clinical and surgical problems. A clear distinction in operative risk must be made. Watkins and Gross[34] and Lewis and associates[35] have recently suggested classifications of atrial defects on the basis of the anatomic location. These, however, appear to us unnecessarily elaborate. The anatomic factor of real importance is the presence or absence of a rim of septal tissue forming the inferior border of the defect above the atrioventricular values. High lesions with aberrant veins entering the right auricle, multiple defects, those with and without posterior, superior, or anterior septum, large ones or those of medium size, all can be satisfactorily and completely closed by direct

suture at a low risk, provided there is present an inferior rim. Successful closure with survival of primum-type lesions, on the other hand, is rarely reported, and in our hands, in four patients, was never accomplished.

Thus, it is imperative and of more than academic interest to anticipate prior to surgery the type of atrial septal defect that may be encountered. Clinically, patients with primum lesions tend to have larger hearts and to become symptomatic earlier than those with the secundum type. Physical examination may reveal findings suggesting the presence of mitral or tricuspid insufficiency, particularly the systolic murmur of insufficiency; the electrocardiogram frequently reveals the presence of left axis deviation and evidence of left ventricular enlargement and thus differs strikingly from the rather characteristic tracing of the secundum type of defect. In our series, although these patients did not present a pathognomonic clinical picture, these features differed sufficiently from the typical findings of the secundum defect that the presence of an ostium primum was considered probable preoperatively in three of these, and this proved to be the case at exploration.

Selection of Patients for Closure.—The establishment of criteria for the selection of patients for closure of an atrial septal defect remains a difficult problem. The great variability in the natural history of patients with this defect has long been recognized. An occasional patient over 70 years of age has been reported with greatly increased pulmonary artery blood flow, a normal pulmonary vascular resistance, and presenting but minimal symptoms. On the other hand, the majority of patients with this disease will develop a decrease in their pulmonary artery blood flow, associated with progressive disability in mid-adult life, and evaluation will establish an increase in the pulmonary vascular resistance and pulmonary hypertension. The development and propagation of occlusive changes within the smaller and, later, the larger pulmonary arteries, we believe to be the process giving rise to these changes which limit the natural history of this disease. However, at the present time, there is no knowledge as to when or why these changes which result in obliterative vascular disease of the pulmonary bed will occur.

TABLE I. PATIENTS OPERATED UPON FOR CLOSURE OF ATRIAL SEPTAL DEFECT BY OPEN TECHNIQUE

	PATIENTS	DEATHS
A. Pure Defect		
Primum lesions	3	2
Secundum lesions	21	2
B. Multiple Defects		
Secundum lesion plus:		
a. pulmonary stenosis	2	1
b. aberrant veins to superior vena cava	1	–
c. patent ductus arteriosus	1	–
d. ventricular septal defect	1	1
Totals	29	6

Evaluation of the 27 patients with secundum lesions operated upon to date reveals that there were 14 below the age of 15, 7 between the ages of 15 and 30 years, and 6 patients over 30 years of age. There have been 4 deaths in this

series. There were no significant differences in the values of the pulmonary vascular resistance in these three groups. Actually, the average resistance in the patients below 15 years of age was the highest. There was, however, considerable difference in the heart size. In all the patients over 30 years of age, the heart was greatly enlarged.

A great deal of importance has recently been placed upon the value of the pulmonary vascular resistance in the selection of patients for closure of their atrial septal defects. Certainly, a patient having a high pulmonary artery pressure and a pulmonary flow less than twice the systemic, and thus high pulmonary vascular resistance, would not represent a good candidate for this procedure. On the other hand, we do not believe that a definite value for pulmonary vascular resistance can be set above which closure of an atrial septal defect is contraindicated. There are so many variables which affect the values employed in determining pulmonary vascular resistance that this determination at best is but a rough index to the changes within the pulmonary vascular bed. Moreover, we found no real correlation between the symptomatology of the patients in our operative series and the computed value for the pulmonary vascular resistance. It cannot be denied, however, that adult patients with enlarged hearts and pulmonary resistances over 300 dynes per second per centimeter –5, present a much greater risk than the young patient with minimal symptoms, a large pulmonary blood flow, and low pulmonary resistance. Such a patient with only a moderately enlarged heart still has a considerable growth period ahead and after closure of the defect, may have a relatively normal cardiovascular system upon the attainment of full growth.

Thus, while it is a matter of grave concern to advise operation in a young, symptomless patient, it is this patient who stands to gain the most from the procedure. The patient over 30 years of age with a greatly enlarged heart and an elevated pulmonary vascular resistance usually has incapacitating symptoms and is definitely in need of help; however, it must be realized that operation upon patients in this group will be attended by a significant mortality and it is certainly not clear, as yet, the degree of improvement that they may expect following successful closure of the defect.

III. SURGICAL TECHNIQUE

The details of the method we have employed has been described elsewhere.[29] In essence, it consists of reducing the body temperature to approximately 26° C., as measured by an inlying rectal thermocouple, by immersion in ice water. The bilateral transverse sternal-cutting incision is placed in the fourth intercostal space. Tapes are placed around the superior vena cava proximal to the azygos vein, and around the inferior vena cava intrapericardially, avoiding injury to the phrenic nerve during both maneuvers. The pericardium is widely opened. After inspection and palpation, stay sutures are placed in the right auricle, a special noncrushing clamp* is applied, and a vertical incision well posterior is made over the clamp. Traction on the tapes now induces inflow occlusion. After thirty seconds, a noncrushing clamp is placed across the aorta 1 inch from the heart and 0.8 to 1.5 ml. of 1:4,000 neostigmine is injected between heart and clamp so as to perfuse the coronary

*Made to our specifications by Geo. P. Pilling & Son Co., Philadelphia, Pa.

arteries. At about one minute, the aortic clamp is removed and a second clamp is placed across the base of both great vessels, occluding the ostia of the coronary arteries. The auricular clamp is now removed and the auricle opened.

The intracardiac procedure is now performed. Visualization of the defect or defects and the point of entrance of pulmonary veins establishes the technical problem. Finger calibration of the size of the mitral valve is made through the defect. The lesion is now closed by suture. Whereas we initially used interrupted mattress sutures, we now use a continuous suture of 00 silk on a half-curved atraumatic needle. A mattress suture is placed

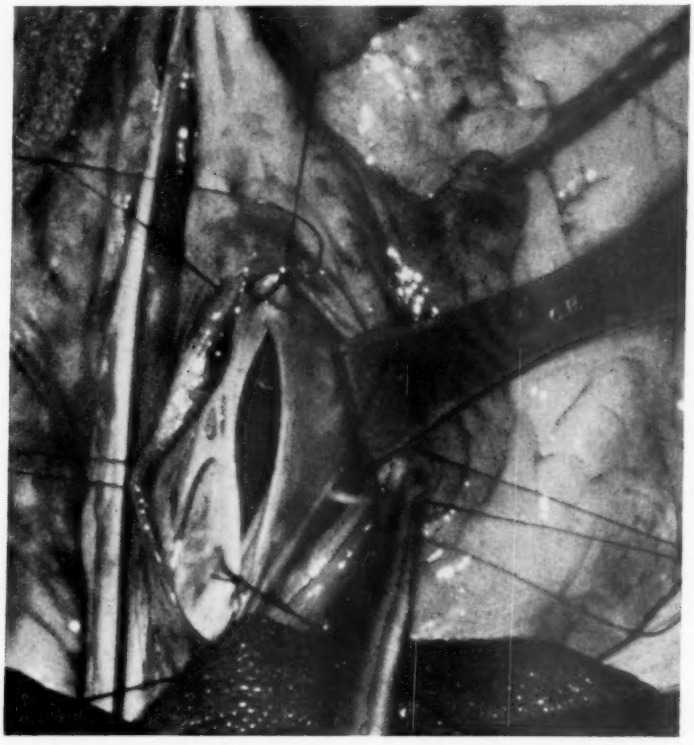

Fig. 1.—An operative photograph showing the process of repair of a secundum-type atrial septal defect. The lumen of right auricle is exposed by the retractor. Through the slitlike defect, a pool of blood in the bottom of the left auricle is seen. A mattress suture has been placed at the lower end and is held by an assistant. A second mattress suture has been placed at the upper end of the defect, which will now be run down as a continuous suture.

at the lower end and tied. A second mattress suture is placed at the upper end. This suture is now run with a continuous over-and-over stitch down to the lower end. A Kelly clamp is placed through the defect at this point to hold it open while the left heart is filled with Ringer's solution to evacuate all air. The running suture is now tied under water to the initial mattress stitch and the ends cut. The defect is thus tightly closed, and coronary air embolism avoided.

Emergence from the heart is accomplished by further flooding the right heart with Ringer's solution and by allowing blood to enter from the superior vena cava as the non-crushing clamp is reapplied to the auricular wall. The outflow clamp is rapidly removed, and the inferior vena cava released. After the heartbeat has strengthened, the myocardium has become pink and circulation is satisfactory, the incision in the auricle wall is closed with mattress sutures of 3-0 silk. The pericardium is loosely closed, bilateral chest tubes are placed, and the incision is closed with wire sutures to the sternum and silk sutures to the muscle layers and skin.

During the entire procedure, hyperventilation to help prevent ventricular fibrillation is employed except during circulatory occlusion when no respiration at all is necessary. Before occlusion a blood pH is determined, and a figure of 7.5 or higher is sought and achieved before occlusion is performed. Blood loss is replaced at once, volume for volume, to the best of our ability to measure and estimate. Blood volumes by the dye method are determined before and about two hours after the procedure to help in this evaluation. Diathermy, with the coils placed around the pelvis, is turned on intermittently immediately following the completion of the intracardiac procedure in order to warm the patient to a point where the arterial blood pressure is at a systolic level of 100 mm. Hg or greater before the thoracotomy is closed, in order that any untied vessels, unrecognized in the dissection during the hypotension of hypothermia, may manifest themselves. The patient is warmed to a temperature of about 35° C., at which time spontaneous respirations are adequate. He is removed to the recovery room and the temperature is allowed to rise slowly without interference during the next several hours.

Fig. 2.—This photograph reveals the continuous suture approaching the lower mattress suture. One more bite will be taken. The left auricle will be filled with Ringer's solution and the two sutures tied. The field has remained dry throughout the closure.

The management of cardiac irregularities occurring during any portion of a procedure during hypothermia has been discussed in previous communications.[29, 36-39] Of the patients presented in this report, 5 entered ventricular fibrillation. Two of these occurred just as the patient was removed from the tub and placed on the table. Immediate thoracotomy was performed and intermittent manual cardiac compression instituted. One heart was restored to a regular beat by the use of potassium chloride, the other by electric shock. Operation was then completed and both these patients are alive and well. The third patient had both pulmonary valvular stenosis and atrial septal defect. The valvular portion of the procedure was carried out with circulatory occlusion via the pulmonary artery in the fashion we have described elsewhere.[40] Since the heart appeared to have tolerated this well, stay sutures were started in the auricle. This manipulation of the heart precipitated ventricular fibrillation which was reverted to a normal rhythm with difficulty after a prolonged period of effort. The patient's incision was therefore rapidly closed and an attempt was made to warm him. However, unrecognized intrathoracic hemorrhage occurred which two hours later resulted

in shock and cardiac arrest from which he did not recover. This mortality is included in the present group because the patient appeared to have tolerated the pulmonary valvular portion of the procedure sufficiently well so that, had we not begun on the auricular maneuver, fibrillation would probably not have occurred and the patient would have survived. The other 2 patients, both with primum-type lesions, fibrillated immediately after the return of circulation. These patients were early in our series, before we began the use of neostigmine. One of these could not be converted; the other recovered but died twenty hours later of heart failure.

Thus, of the 5 hearts in this group which entered ventricular fibrillation, all but 1 were restored to normal rhythm and 2 of the patients are long-term survivors with complete cure of their lesions.

Two other deaths were caused, we believe, by progressive thrombosis of the pulmonary arterial tree. One of these occurred on the fourth day, the other on the eighth day following surgery. Both patients were doing extremely well only to enter a rapid downhill course which resulted in death in twelve to thirty-six hours. At autopsy, the lungs showed widespread but patchy areas of infarction varying from lobular to lobar in size. In one, the pre-mortem thrombosis of the vessels was recognized. In addition, we have seen another patient who underwent an identical clinical course and whose lungs showed identical pathology without operation having been performed. Anyone who has seen the massive dilatation of the pulmonary arterial tree associated with the large pulmonary blood flow of atrial septal defect must have pondered the ultimate fate of such enlarged vessels when the blood flow is suddenly reduced to one-third or less of its previous volume. We have already described our concept that gradual progressive thrombotic obliteration of the peripheral ramifications of this arterial tree is the main cause of the rising pulmonary resistance seen in the later stages of the unaltered course of the disease. In the same fashion, more rapid extension of the thrombosis into large vessels would be favored by the sluggish flow induced by a sudden reduction in volume flow without a simultaneous and equal reduction in the vascular cross-sectional area. We are so impressed with this phenomenon that we intend to use anticoagulants beginning immediately after surgery in patients with massive enlargement of the pulmonary arterial tree.

The remaining death resulted from postoperative hemorrhage from an internal mammary artery injured during closure which was not recognized until shock occurred. The patient entered a period of oliguria and died suddenly just as she was disconnected from the artificial kidney on the twelfth day.

Associated cardiac abnormalities were present in 7 patients in this series. In 4, pulmonary valvular stenosis (trilogy of Fallot), and, in 1 each, aberrant pulmonary veins entering superior vena cava, patent ductus arteriosus, and ventricular septal defect were also present. Mitral stenosis was not encountered. It is often true, of course, that the mitral valve is small, proportionate to the relatively small size of the entire left heart and systemic circulation. If measured on the autopsy table it would no doubt be found to be considerably smaller than normal. However, no fusion of commissures, no thickening of leaflets, or shortening of chordae tendinae were felt in this group of patients. True Lutembacher's syndrome, we think, must be very rare. Aberrant veins entering the right auricle present no problem; they can be incorporated into the lumen of the left auricle by placing the suture line to their right. If they enter the superior vena cava well above the auricle, however, a special problem is created and transplantation must be considered. Patent ductus arteriosus in association with atrial septal defect adds an additional left-to-right shunt which can be easily closed at the same procedure. We think the combination must be quite rare. The associated ventricular septal defect was a small lesion near the apical portion of the septum. Its significance in the widespread pulmonary arterial thrombosis which killed this patient on the eighth postoperative day is questionable.

The largest group with associated lesions is comprised of those patients with the trilogy of Fallot. Of our 4 patients, 2 were cyanotic, the atrial shunt being right-to-left, while 2 were acyanotic, with the shunt being left-to-right. Our results in these individuals have

led us to the opinion that there are special, important considerations in the treatment of this syndrome. The disease consists of 2 defects, both of which are completely amenable to repair by our open techniques, yet our results have left much to be desired. One patient who died when repair of both lesions was attempted at a single stage has been described. Another patient, an infant, died about eighteen hours following relief of valvular stenosis, a sudden death which remained unexplained following autopsy. Both of these patients were cyanotic. The 2 noncyanotic patients both survived pulmonary valvuloplasty as a first stage procedure. Their hearts increased in size and it was presumed that the left-to-right atrial shunt increased in volume. One of these patients subsequently has undergone successful repair of the septal defect while the other is awaiting surgery. Thus, of these 4 patients, the 2 with cyanosis are dead, while the other 2 live, 1 completely cured, the other needing repair of the atrial defect. The ramifications of this experience will comprise the subject of a future report.

In our opinion, there is only one worth-while result of surgery in atrial septal defect, that is, complete closure. It is remarkable how little is accomplished by 80 per cent closure of these defects. This opinion is documented by our experience in one extremely interesting patient (W. G.). This 6-year-old boy underwent operation by one of us (H. S.) in 1952 for closure of his large atrial septal defect using the method we had developed which sutured in place a button of polyethylene film by a blind technique. This method has been previously described.[13] At the time of the operation, it was the surgeon's opinion that the 2.5 cm. button was in good position and that it was of sufficient size to occlude most of the defect, leaving only a crescent-shaped opening along the posteroinferior border of the prosthesis. The degree of closure was estimated at 80 per cent. Following convalescence, it was the mother's opinion that the child was improved in his exercise tolerance, but repeat catheterization revealed essentially no change in the volume of the left-to-right shunt. The heart remained grossly enlarged. Accordingly, in 1954, he was re-explored during hypothermia and the septum visualized. The situation was seen to be exactly as expected. The button was encased in a gray, glistening membrane and was attached firmly to atrial septum throughout the upper two-thirds of its circumference. A crescent-shaped defect remained along the posterior-inferior portion. Again, the degree of closure effected by the first operation was estimated to have been about 80 per cent. Using interrupted mattress sutures, the remaining defect was closed by suturing the residual rim to the button, which was left in place. Recovery was again uneventful, and repeat catheterization revealed that the defect was finally completely closed. The original, proved 80 per cent closure had not significantly changed his shunt!

Another child in this current series (J. U.) had closure of a large secundum defect with interrupted sutures. Postoperatively, the shunt remained large (Table II). At a second operation a residual gap at the very lower end of the defect was closed by two additional sutures. The previous closure was estimated to have been at least 85 per cent.

In the light of these experiences, we have shifted to the continuous suture technique, and to date are satisfied with the complete closure achieved by this method. The importance both of complete closure and of postoperative objective evaluation, we feel, are highlighted by these experiences.

IV. RESULTS

In the past three years, 100 patients having atrial septal defects have been evaluated and, of this number, 30 have been selected for closure of their defect by direct vision during hypothermia. Three had primum lesions; the remainder were of the secundum type, and 5 of these were associated with an additional cardiac anomaly. There were 24 survivors in the group. Postoperative catheterization studies have been completed on 21 of these, and the results are tabulated in Table II.

TABLE II. RESULTS OF OPEN CLOSURE OF SECUNDUM LESIONS (BASED ON CATHETERIZATION DATA)

PATIENT	PULMONARY ARTERY PRESSURES		RATIO OF SYSTEMIC TO PULMONARY INDICES		COMMENTS
	PREOP.	POSTOP.	PREOP.	POSTOP.	
			A. Pure Lesions		
G. H.	100/30	30/17	3.1	1.0	Closed
C. C.	27/16	20/10	4.7	1.0	Closed
A. C.	20/4	29/13	5.9	1.0	Closed
M. E.	56/12	24/3	2.1	1.0	Closed
W. F.	38/15	30/18	2.4	1.0	Closed
D. G.	26/4(RV)	15/7	3.3	1.0	Closed
C. G.	49/18	35/11	2.3	1.0	Closed
R. C.	30/11	38/17	2.3	1.0	Closed
J. G.	40/5(RV)	44/13	2.3	1.0	Closed
A. M.	38/13	30/12	3.5	1.0	Closed
N. C.	30/15	30/13	3.9	1.3	Only minimal shunt remains
C. P.	28/13	22/10	3.4	1.0	Closed
D. D.	43/25	20/10	7.2	1.0	Closed
J. U.	36/0(RV)	-----	2.9	2.6	No significant closure (85%) Closed at second Procedure
S. K.	30/16	23/14	3.5	1.0	Closed
J. C.	35/14	41/18	2.2	1.0	Closed
T. G.	39/12	26/9	3.4	1.0	Closed
			B. Associated Anomalies		
R. G.	50/36	38/12	3.3	1.2	Anomalous connections of PVV to SVC; closed
P. S.	33/10	27/3	2.6	1.0	Valvular pulmonic stenosis; closed
E. A.	81/46	38/18	4.5	1.0	Patent ductus arteriosus; closed

These data reveal essentially complete closure in all but 2 patients. In 1 this was not unexpected. As mentioned, these were 2 patients with massive defects, which were closed by interrupted suture. Since that time, after adoption of the continuous suture, we have had no patients with residual shunts.

The pulmonary pressure was within normal limits or but slightly elevated in the majority of these patients and revealed no significant changes following surgery. Patients showing definite pulmonary hypertension prior to closure of their defect, however, all revealed a postoperative decrease in the pulmonary artery blood pressure. Thus, with the great reduction in pulmonary artery blood flow and the minimal changes in the pulmonary artery pressure that occurred in most of these patients, there was a rise in pulmonary artery resistance following closure of the defect.

Dyspnea and ease of fatigue was a prominent feature of the preoperative course in several of these patients, and there has been a great amelioration of these symptoms postoperatively. All patients who were symptomatic prior to operation have returned to full activity six months following surgery.

V. DISCUSSION

We recognize a profound difference in the physiologic and anatomic pathology between primum and secundum lesions. We make a strong plea to those who report on this disease in the future to separate these entities and to discuss them individually as Bailey and associates[42] have done, so that comparative evaluations can be meaningful.

In addition, the presence of associated cardiac anomalies basically alter the natural course and operative risk of the disease. These, too, should be distinguished. The presence of pulmonary valvular stenosis is particularly pertinent in this regard.

For these reasons we present Table III with considerable hesitancy lest too much emphasis be placed upon it. The groups tabulated vary considerably in the number of different types of lesions included; the operative indications may well have been different; and, in addition, since they all represent the beginning experience of the various surgeons with new techniques, more recent application is probably considerably improved. Thus, we feel confident that the lessons we have learned should enable us to realize even more fully the potentials of our technique in the future. Nonetheless, it is the only method available to us at this time which attempts to document our belief in the superiority of the visual method of repair of this lesion.

TABLE III. EVIDENCE OF CLOSURE IN REPORTED SERIES WITH VARIOUS PROCEDURES

AUTHOR	NO. OF PATIENTS	PROVED CURE	MODERATE SUCCESS	FAILURE	UNKNOWN	DEAD
A. Closed Techniques						
Gross and Watkins[41] (1953)	15	3	–	1	3	8
Bailey[42] (1954)	46	15	1	5	13	14
Kirklin[43] (1955)	12	4	2	1	5	1
Muller[27] (1955)	18	8	1	1	5	3
B. Direct-Vision Suture						
Lewis[35] (1955)	36	10	2	–	17	7
This series (1955)	30	19	1	1	3	6

We believe this evidence suggests that open operation during hypothermia can be performed with a degree of safety which compares favorably with closed techniques. The high incidence of complete cure of secundum lesions by this method is particularly gratifying.

VI. SUMMARY AND CONCLUSIONS

1. A series of 30 patients with atrial septal defects who underwent operation for the purpose of closure of these defects under direct vision during hypothermia is analyzed.

2. Of 27 patients having secundum type lesions, 5 had associated cardiac anomalies exclusive of aberrant pulmonary veins entering right auricle, and 23 survived.

3. Of 3 patients with primum lesions, only 1 survived, uncured.

4. Postoperative catheterization data are available on 21 patients, and reveal closure of the defect in all but 2.

5. Total closure of the anatomic defect is essential if the disordered physiology is to be cured. Eighty per cent closure will have little or no effect upon the volume of the shunt.

6. For the direct-vision closure of very large defects, a continous suture is preferable to interrupted ones if complete closure is to be achieved.

7. Direct suture during hypothermia of secundum type atrial septal defects is extremely effective and can be carried out at a low risk.

8. Patients with secundum defects who demonstrate a large left-to-right shunt should be offered operation at an early age, before they begin to have excessive dilatation of the pulmonary arterial tree, cardiac enlargement, or progressive elevation of pulmonary resistance, since the risk of operation is quite low before these changes occur. Patients with extremely high pulmonary resistance and minimal or reversed shunts have passed the stage of surgical benefit.

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THE DIVERTICULUM APPROACH TO THE CHAMBERS OF THE HEART AND GREAT VESSELS

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THE diverticulum approach to the heart chambers and great vessels is a technique whereby the normal confines of these structures are extended by means of a closed tube or pouch of rubber, plastic, fabric, or tissue. The attachment of this extension is effected by direct suture to the wall, suture to an incision in the wall, or the application of a specially designed metal "ring clamp." The manipulations which can be carried out through these tubes are blind and are made by instruments alone or by finger and instruments.

Historically, the idea of using a closed pouch or diverticulum to approach a chamber or cavity of the body probably originated with Galen. Quoting from a translation of one of his early experiments to determine if air was present between the lung and chest wall during life:

> A small incision was made in the skin of the thorax and to this the mouth of a bag or bladder was carefully stitched, and the juncture then secured with some adhesive material such as wax to make it air-tight. A round-handled scalpel was then pushed through the bladder and the opening thus made in the bladder was securely tied around the handle. The point of the scalpel was then driven through the parietal pleura and so continuity was established between the intrathoracic cavity and the air in the bladder.[13]

The instrumental and digital approach to the chambers of the heart and great vessels through a closed artificial extension was evolved independently by several workers using certain basically different techniques. One of these was that described by Söndergaard in the Transactions of the twenty-fourth Meeting of the Northern Surgical Association in Helsinki, Finland, in June, 1949. This technique consisted of using a rubber tube extension of the heart chamber or great vessel, attached to a specially designed "ring clamp." No sutures were used to hold the rubber extension in place, no digital exploration was possible through the rubber extension, and no heparinization of the blood in the rubber tube was reported. This technique was used on the pulmonary artery of a human being for the first time by Andersen in 1951,[1] and by Söndergaard in 1952.[14]

The technique developed independently by Glenn in 1952[6] was inspired by the needs of a small infant with a large ventricular septal defect. It had been

Aided by a grant from the United States Public Health Service H 851 C4, National Institutes of Health.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

shown by Gross[11] in his ingenious experiments on an open artificial (rubber) extension to the right auricle that the suture line became "waterproof," and it occurred to us that perhaps a closed artificial extension to the high pressure area of the heart and great vessels could be employed temporarily, provided the suture line could be maintained free of leakage. When a closed rubber extension was applied to the experimental animal the suture line was found to be "waterproof" on all chambers of the heart and the pulmonary artery, but on the aorta the force of the arterial blood pressure directed into the rubber extension caused frequent leakage at the suture line, and distention of the rubber.

Bailey and associates[4] and Swann and co-workers[16] have been successful in the aortic approach to the aortic valve in the patient using a diverticulum, or what Swann would prefer to call an "operative tunnel," made from a flap of pericardium or a plastic-lined fabric sewed directly to an incision in the aortic wall. Swann reported that they had also used an artificial extension to the pulmonary artery and to the right ventricle. Recently, Pearl and associates[12] have independently reported experiments on an instrumental approach to the aortic valve through a diverticulum of latex sutured to an incision in the aorta.

The first application of the diverticulum to the heart proper is believed to have been by Glenn, Shannon, and Turk on Dec. 17, 1953,[9] in a patient with mitral stenosis. This patient is living and continues improved approximately eighteen months after operation. This first clinical use followed more than 100 trials in the experimental animal. The simplicity of this technique and the possibility of a prolonged digital examination of the intracardiac lesion resulted in the application of the diverticulum to other chambers of the heart and great vessels. It is the purpose of the present communication to review briefly the use of the diverticulum approach to the heart chambers and great vessels from our own personal experience, and to call attention to certain technical considerations in the use of this technique in the patient.

MATERIALS

The Diverticulum.—The diverticulum used by us currently is made either of latex[6] or of Orlon fabric. A simple latex diverticulum which is sufficiently thin to be easily manageable is probably not safe where the blood pressure is in excess of 50 mm. Hg. The latex diverticulum is now made in three sizes dependent upon the length of the base. Three extensions are present: the largest extension is for the finger, the middle for the insertion of an instrument, and the smallest for the infusion of a drip of heparin solution.

The fabric diverticulum suggested by one of us (T. O. G.) is made from du Pont 100 per cent Orlon cloth. The cloth is of a plain taffetta weave, 100 denier, warp and filling count 80 by 72. The diverticulum and extensions are constructed from a flat piece of cloth with a double seam of nylon. In addition, a single, circular line of sewing-machine stitch of nylon thread is placed at the base of the diverticulum for the purpose of reinforcement at the point of suture of the fabric to the tissue (Fig. 1).

Method of Entering and Closing the Heart Chambers and Great Vessels.—Both the rubber and the Orlon diverticulums are sutured to the tissues in an essentially similar manner. At both ends of the incision in the tissue, or just beyond the proposed limits of the incision

if none is to be made before the diverticulum is sewn into place, an over-and-over suture of 4-0 braided arterial silk is placed, including first one side and then the other of the corner of the incision, and in a similar manner the two sides of the corner of the diverticulum. With the base of the diverticulum fixed at both ends, the interrupted sutures at either end are joined by an over-and-over continuous suture. Fairly generous bites of tissue should be taken. On the rubber diverticulum there is a bead of rubber at the base, and this should be rolled over once prior to placing the suture through the rubber just behind it. With the Orlon diverticulum, the suture in the fabric is placed just behind the reinforcing stitch of nylon. Where the Orlon is sutured to the aorta, it is wise to take an additional over-and-over suture on both sides of the corner sutures. These sutures then, rather than the corner ones, are joined together by continuing each to the middle of the base of the diverticulum.

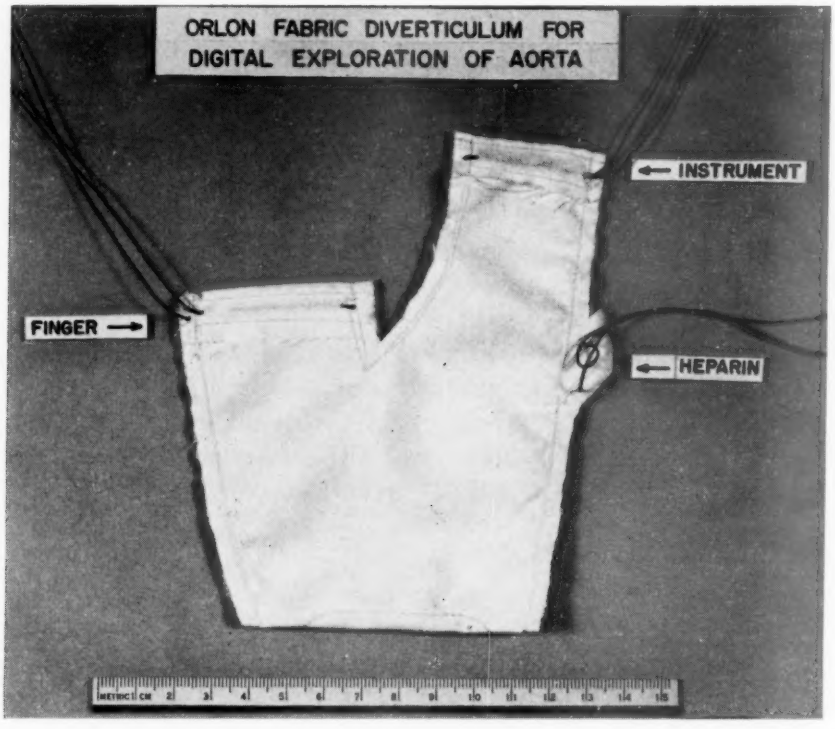


Fig. 1.—After the 4 cm. incision is made in the aorta, the bottom of the Orlon diverticulum is cut open and the edges sutured to the incision. Note the reinforced nylon stitch that encircles the bottom of the diverticulum (near the scale). The opening in the Orlon is made within the area enclosed in this stitch.

The diverticulum is sewn to an incision previously made in the heart or vessel wall, or directly to the heart wall, depending upon whether one is able to secure a portion of the wall and lumen of the structure in an occluding noncrushing clamp. Clamps of the Satinsky-Glover type are well suited to this function and we have four pairs of these clamps available for use, with jaws of varying lengths. Included among them is the extraheavy clamp[3] for use on the aorta. When a diverticulum is to be sewn to the ventricle or to a tightly distended auricle, it is not possible to pick up a portion of the wall and lumen safely in a clamp. Therefore, in either case, the diverticulum is sutured directly to the intact wall. The incision into the lumen of the chamber is made after the suturing is completed. A small incision is made at first, to detect and correct with additional interrupted over-and-over sutures of 4-0 silk any leakage at the juncture of the diverticulum with the heart. The DeBakey-Beaver long-handled knife is used to make this incision through the diverticulum

because the back of the detachable blade is protected and does not catch on the wall of the diverticulum. One must take care not to extend the incision beyond the limits of the diverticulum.

Closure of the incision in the heart or great vessel is made after reapplication of the noncrushing clamp below the diverticulum with a double row, interrupted at both ends of the incision, of an over-and-over suture of 4-0 silk. Additional interrupted over-and-over or mattress sutures are sometimes indicated. The use of the noncrushing clamp in the closure of the incision in the heart or great vessels is the preferable technique, and it should be employed whenever possible. However, sometimes, as on the ventricle or with only a narrow space available on the auricle, it is not possible to apply the clamp, and closure must be accomplished by mattress sutures or by the use of temporary parallel bars of polyethylene adapted to a modified Bethune clamp.[9]

Clotting and Anticoagulants.—Clot formation in a rubber or Orlon diverticulum will promptly occur unless measures are taken to prevent it. Clotting appears to be more prompt and extensive in the presence of Orlon.

In the case of the rubber diverticulum, a small amount of fresh blood is allowed to run into the diverticulum and to clot around the suture line. Excess clot is then removed by suction through one of the extensions, the diverticulum is thoroughly rinsed with saline solution, and a heparin drip is started. A solution of aqueous heparin (20 mg.) in normal saline solution (100 c.c.) is allowed to drip into the diverticulum at 10 to 15 drops per minute. At this slow rate, we have been able to prevent clot formation in the rubber diverticulum in most cases, without evidence of a local or systemic bleeding tendency. The base of the rubber diverticulum or of any of the extensions has been repeatedly clamped without, so far as we know, any peripheral clot embolization.

Periodically, the finger should be removed from the heart chamber or pulmonary artery and the diverticulum clamped just above the suture line. The finger is then removed from the diverticulum extension. Any fibrin formation that may have formed on the glove of the exploring finger will in this way be peeled off into the clamped-off portion of the diverticulum and not be discharged in the blood stream. The inside of the diverticulum is carefully inspected for clots which, if present, are removed.

With the use of the Orlon fabric diverticulum, the wall must be made impermeable by allowing the blood to clot within the interstices of the cloth. This is accomplished on the aorta, the only vascular structure on which we have used the Orlon diverticulum, by temporarily releasing slightly the noncrushing clamp after suture of the Orlon to the edges of the incision, allowing some blood to enter the diverticulum and bathe the cloth. All parts of the cloth should be submerged in the fresh blood bath. The clamp is then promptly retightened and after two minutes is gently and partially released for a moment. This is repeated again two minutes later. Usually by this time the cloth is almost entirely impervious to blood. Care must be taken that the clamped-off open ends of the fabric diverticulum are also allowed to be bathed in blood. It is important that the clamp on the aorta not be removed completely until the fabric is shown to be leakproof and all gross clot is removed from the inside of the diverticulum. After this has been done, the diverticulum is filled with saline solution and the heparin drip is begun. The clamp on the aorta is then slowly released. Leakage at the suture line has been very unusual with the use of Orlon fabric, but should it occur, the clamp should be reapplied and a suture taken to correct it. If bleeding from the fabric recurs, it will usually stop promptly if the heparin drip is discontinued momentarily. The exploring finger should be constantly alert to the presence of large soft clots and if any are detected, at any time, the aorta should be reclamped and the diverticulum cleaned out. It is probably not safe to clamp the base of the diverticulum under these circumstances, because the clot may be pushed off into the blood stream.

Instruments.—Specially designed instruments are sometimes used with the diverticulum. In general, the instruments that require a moving jaw employ parallel bars, as with the standard pituitary forceps. In order to use the instrument as an exploring "finger" or

TABLE I

CASE NO.	AGE (YEARS)	SEX	PREOPERATIVE DIAGNOSIS	DATE	TREATMENT	DIVERTICULUM		COMMENTS
						SITE	DURATION (MINUTES)	
38-67-14	52	F	Mitral stenosis	12/17/53	Valvulotomy	LA	110	Improved 18 months postop.
39-50-37	8/12	F	IASD, IVSD	3/13/54	Exploratory	RV	100	Died postop.; tricuspid atresia and no pulmonary stenosis found at post-mortem
41-01-03	36	F	Mitral stenosis	6/26/54	Valvulotomy	LA	85	Instrumental valvulotomy made possible
41-00-00	43	F	IASD, mitral stenosis, double SVC, RA insertion of rt. upper pulmonary veins	7/20/54	Valvulotomy, atriopexy, lig. rt. SVC	RA	130	At 10 months is apparently cured; cyanotic preop.
38-68-32	20	M	Pulmonary stenosis	9/23/54	Valvulotomy	PA	70	Valve cut on one side only
40-81-69	39	F	IASD, Mitral stenosis	12/ 9/54	Valvulotomy	RA	60	Extensive adhesive pericarditis; septum primum defect could not be closed; at 6 months, improved
42-10-48	46	F	Pulmonary stenosis	2/ 1/55	Valvulotomy	PA	80	Did well for 6 days; died with acute arrhythmia and intense cyanosis
C34877	32	M	Mitral insufficiency	2/11/55	Vertical graft	LA	90	Appeared improved at 5 months postop.
C44340	43	F	IASD	3/12/55	Atriopexy	RA	145	Improved; cyanotic preop.
B15529	44	F	IASD	4/28/55	Atriopexy	RA	105	Improved
A50540	18	F	Pulmonary stenosis	4/12/55	Valvulotomy	PA	70	Cyanotic preop.; moderate cyanosis still present on discharge
41-70-83	21	F	Pulmonary stenosis	5/21/55	Valvulotomy	PA	45	No gradient RV-PA immediately postvalvulotomy
42-82-57	28	F	Aortic stenosis	6/ 1/55	Valvulotomy	Aorta	35	Orlon diverticulum

"eye," a metal tube may be attached to the end of the instrument. The other end of the tube is then attached to a woven catheter which in turn attaches to a pressure capsule and oscilloscope (Fig. 2). A needle with a detachable point has also been found useful.[7]

The success or failure of an operative procedure within the heart performed blindly with the finger depends largely upon the ability of the exploring finger to appraise accurately the physiologic and pathologic alterations. We have suggested that an extremely thin glove be made for intracardiac digital exploration.* Since these gloves tear easily, they should probably not be used for any other less delicate purpose. As a result of the operative procedure a small hole may sometimes develop in the tip of the exploring gloved finger.

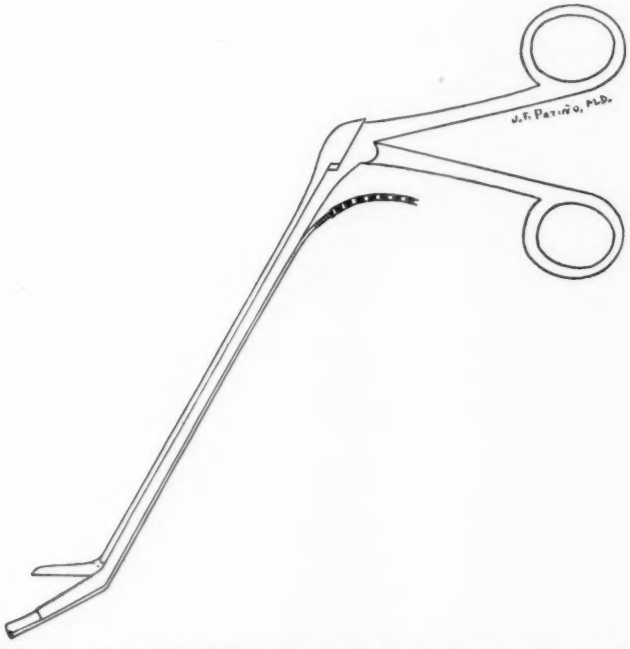


Fig. 2.—Scissors with small metal tube permanently attached to lower blade. Catheter attached to end of the tube near handle is connected with oscilloscope and pressure recorded.

CLINICAL APPLICATION

The diverticulum approach to the chambers of the heart and great vessels has now been used by us in 13 cases. Entry has been made to all chambers of the heart except the left ventricle, and to both great arteries (Table I). A rubber diverticulum has been used in all cases except the one involving the aorta, where the Orlon fabric diverticulum was used.

Right Auricle.—The diverticulum has been used as a method of approach to the right auricle chambers in four patients. Each of the four had an interauricular septal defect. Two had an associated mitral stenosis, and in two there was an associated anomalous entry of the right upper pulmonary veins into the right auricle. Exposure of the right atrium was gained by a transsternal incision in the fourth intercostal space was advocated by Swan.[15] The diverticulum was usually sewn to the edges of an incision in the tip of the right auricle, but in one case it was sutured to an incision in the middle

*Made by Seamless Rubber Co., New Haven, Conn.

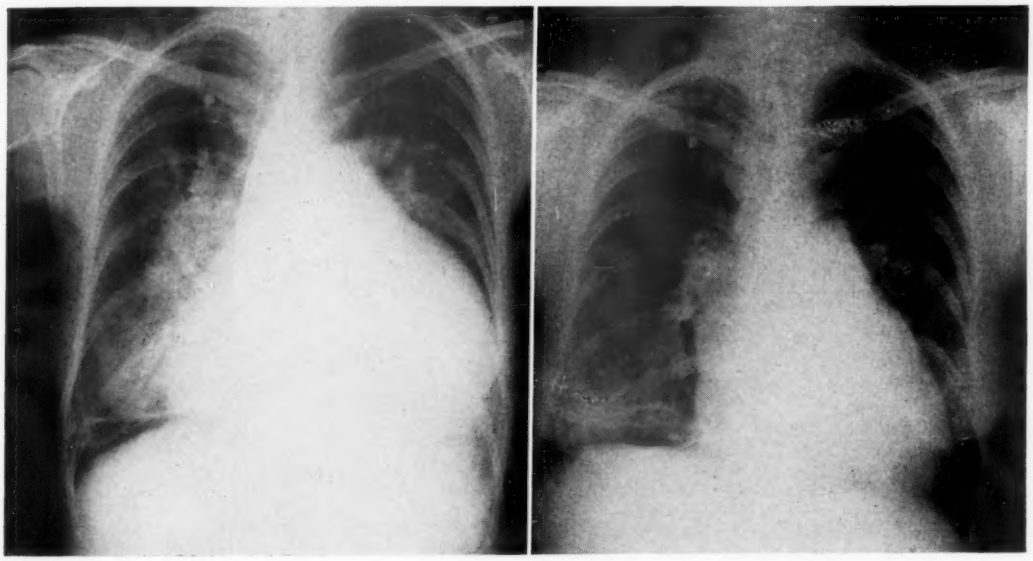
of a greatly enlarged auricle. No problems were encountered with the opening or closing of the incision in the heart. The mitral stenosis was corrected by digital valvulotomy in one patient, and by instrumental valvulotomy in the other. In both cases, the valvulotomy was performed from the right side through the septal defect. In one patient an extensive adhesive pericarditis was present in addition to a large septum primum defect. Closure of this defect could not be accomplished. Six months following relief of the mitral stenosis, the patient is improved. The auricular septal defect was closed in the other three patients of this group by the atrio-septo-pexy technique, with redirection, in two of these, of the pulmonary venous return into the left auricle.[5] All survived and appear to be markedly improved, although for two of them it is too early for final evaluation of the septal closure. Cardiac catheterization has been carried out in one of these patients ten months following operation. The catheterization data presented in Table II would indicate that the septal defect is closed and that there is no elevation of the pulmonary artery pressure. During this postoperative period of ten months she has gained about 35 pounds. An x-ray examination of the chest taken preoperatively may be compared with the chest film taken ten months following operation (Fig. 3).

TABLE II. CARDIAC CATHETERIZATION

CATHETER POSITION	BLOOD OXYGEN CONTENT VOLUME PER CENT		SATURATION PER CENT		PRESSURE Mm. Hg	
	PREOP.	POSTOP.	PREOP.	POSTOP.	PREOP.	POSTOP.
I.V.C.	7.52	11.12	49		20	
R.A.	13.02	10.90	84		28/22–	2/-2
R.A.	13.20	10.85	85			
P.V., rt., high	15.05		97		30/18–	
P.V., rt., low	14.87		95			
P.A.	12.17	11.27	79		84/40	29/11
R.V.	12.66		82		83/20	30/-4
Femoral artery	13.87	16.76	89	94.3	55 (mean)	
Femoral artery after oxygen	14.52	18.85	91	>100.0		

Left Auricle.—The diverticulum was applied to the left auricle in three patients. In none could the approach to the auricular chamber be made through the auricular appendage. Two of these patients had predominant mitral stenosis and one, predominant mitral insufficiency. In one patient, the diverticulum was sutured to an extension of an incision in the auricular appendage to permit a more satisfactory valvulotomy. In the other two patients, the auricular appendage had been used before in the previous operation. The diverticulum was sutured directly to the wall of the auricle in these patients, and an incision made into the auricular chamber through the diverticulum. There was some bleeding from the suture line of the diverticulum in the first of these patients, probably the result of taking too small bites of the myocardium. Both patients represented a problem in closure of the incision in the heart. In one[9] the parallel bars of polyethylene failed to evert the edges of the auricle, and mattress sutures placed beneath the diverticulum were necessary. In the second, a patient with mitral insufficiency and an

enormous, thin-walled auricle, the mattress sutures placed to close the diverticulum incision began to tear the auricle wall as they were tied down. They were therefore abandoned. Fortunately, the pressure in the auricle was lower after repair of the mitral insufficiency with a vertical graft[10] than before, so we were then able to place a long-bladed Satinsky-Glover type clamp beneath the diverticulum and to close the auricle over that. As a result of this experience, we believe that before sewing a diverticulum to the left auricle it is important to search out, by needle puncture if necessary, a relatively thick part of the auricular wall, for if there is increased pressure from within, tearing of the wall might result from attempts to close the incision. All these patients survived the operations and are apparently improved, five to eighteen months following operation.

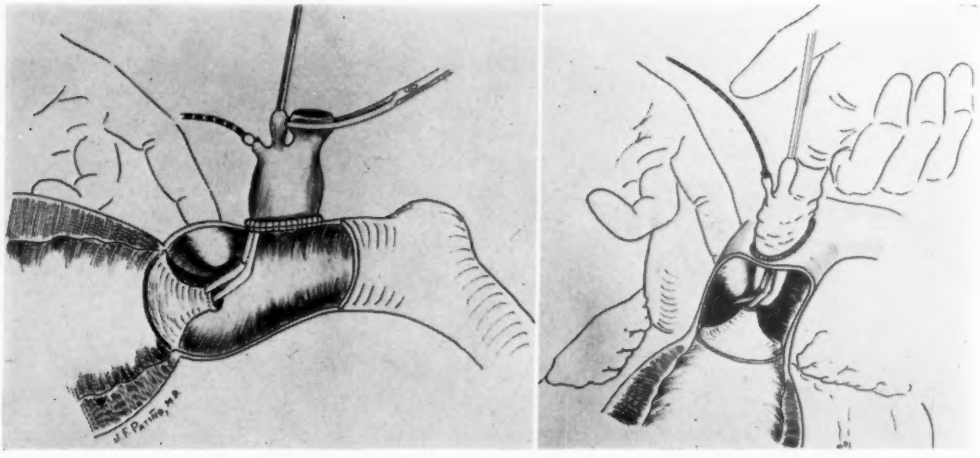


A. *B.*

Fig. 3.—Patient 41-00-00. *A*, Preoperative chest film; *B*, 10 months postoperatively.

Right Ventricle.—In only one patient have we applied the diverticulum to the right ventricle. This patient was an 8-month-old infant who had been hospitalized since the first months of life. Cyanosis had been noted intermittently from birth. Cardiac failure had been present from 10 weeks of age. The electrocardiogram revealed left ventricular hypertrophy. She had gained only 1,500 grams from birth to 8 months of age, and had required oxygen almost continually. Cardiac catheterization revealed a "right ventricular" pressure of 97/0 mm. Hg. An interauricular septal defect was also demonstrated. Preoperatively it was believed that she had a large interventricular septal defect in addition to the auricular defect. The blood flow to the lungs was increased. Because of her progressive worsening condition, the decision was made to explore the right ventricle in the hope that the ventricular defect could be repaired.[17] No difficulty was encountered with the diverticulum approach to the right ventricle or to the closure of the incision. However, where

the diverticulum was sewn in place one small branch of the right coronary artery crossed just beneath one edge of the diverticulum. Although we were able to suture around this branch we unknowingly severed it when we opened the ventricle. This may have contributed to the patient's already poor condition. The coronary vessels are a constant hazard to an operative approach through the ventricular wall. When an attempt was made to place a suture in the margin of the septal defect, the heartbeat developed an atrioventricular dissociation. Further exploration was discontinued. Closure of the ventricular wall was facilitated by the temporary use of parallel bars of polyethylene with a mattress suture attached to a modified Bethune clamp.[9]



A. *B.*

Fig. 4.—*A*, Diverticulum sutured to pulmonary artery and valvulotomy performed with scissors introduced through diverticulum. Left index finger outside of pulmonary artery guides scissors blades into stenotic valve opening. *B*, Pulmonary valvulotomy with right index finger inside pulmonary artery to guide scissors blades into stenotic valve opening.

The patient survived the exploration but died in the early postoperative period. Post-mortem examination revealed large interauricular and interventricular septal defects and an unsuspected tricuspid atresia, and no stenosis of the pulmonary artery. In retrospect, the tricuspid atresia should have been recognized at the time of the right ventriculotomy, but it was not suspected in the presence of a normal pulmonary valve and a preoperative catheterization finding believed to be in the "right ventricle."

Pulmonary Artery.—In four patients, a pulmonary valvulotomy has been performed through a diverticulum sutured to an incision in the pulmonary artery. All patients were adults. The youngest and smallest patient was 18 years, the oldest 46. Three of the patients were moderately to severely cyanotic. The operative incision was made through the fourth intercostal space in each patient, using a long lateral incision. The incision was carried from the sternum in front to the angle of the rib posteriorly. Provision was made for the patient to be promptly turned onto his back in the event that it became necessary to complete the valvulotomy through the ventricular route.

Upon opening the chest, there was a fall in blood pressure noted in each patient, which could be corrected by the intravenous infusion of Levophed. No difficulty was encountered in suturing the diverticulum in place to a 3 cm. incision in the pulmonary artery. Direct palpation of the valve through the diverticulum permitted an accurate assessment of the depth of the sinuses of Valsalva, the fusion of the commissures, and the size and form of the opening in the valve. After the decision had been made where to cut the valve, it was somewhat easier to direct the scissors into the stenosed valve opening with one finger on the outside of the pulmonary artery. Also, with the scissors open, one can palpate with ease the back of the upper blade with the finger on the outside of the pulmonary artery (Fig. 4).

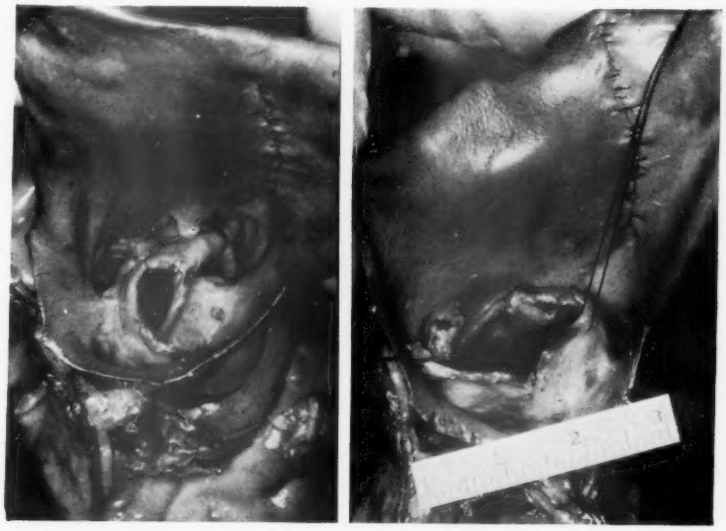
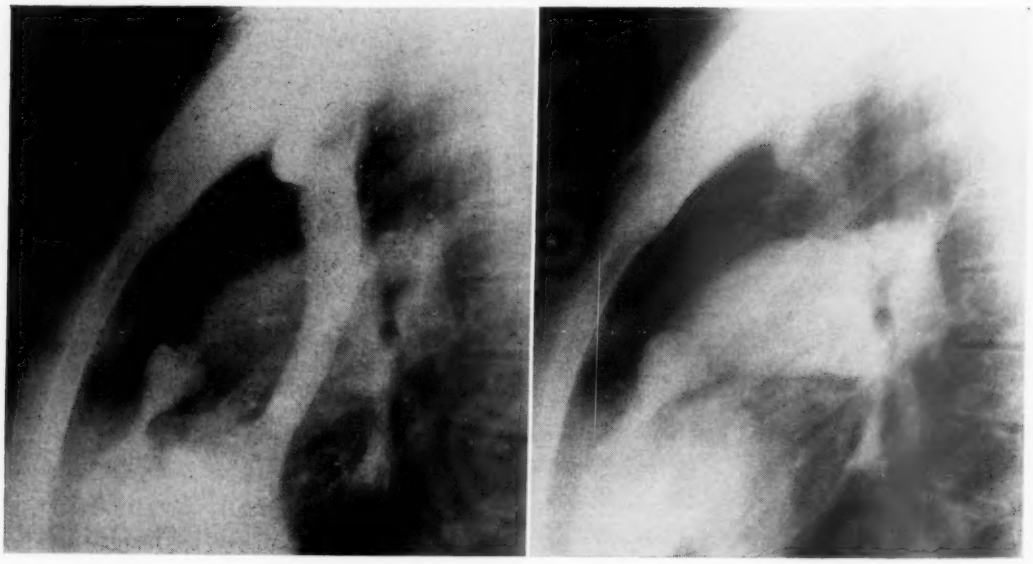


Fig. 5.—Patient 42-10-48. Pulmonary valvulotomy. The valve at the left is shown as it was prior to opening. On the right, the cusps were held apart by sutures exposing the incisions made at operation six days before. No valvular insufficiency was noted in the immediate postoperative period.

It was sometimes difficult to determine when the scissors blade was in the valve orifice. A section of catheter was then attached to the lower blade to guide the blade into the stenosed opening of the valve by oscilloscopic control. This catheter has recently been permanently incorporated into the design of the scissors in the form of a metal tube. Other scissors of varying sizes are sometimes useful (Fig. 2).

In none of these four patients was the opening in the valve prior to valvulotomy sufficiently large to admit the very tip of the finger. In one, the point of the lower scissors blade completely blocked the systolic jet. A wide opening was obtained in each patient, easily admitting the entire finger and permitting the exploration of the right ventricle from above. In two patients of this group, the first and the third, no more than a finger and a half opening in the pulmonary valve was obtained. The first of these patients was recatheterized six months following operation, and showed a right ventricular pressure of 96/0 mm. Hg, compared with 210/0 mm. Hg prior to

operation. We believe that with increased experience, a more satisfactory valvulotomy can be obtained. A widely open valvulotomy was obtained in the other two patients. One of these patients, aged 46, died on the sixth postoperative day coincident with the onset of auricular flutter fibrillation. The valve had been widely opened at operation, as shown in Fig. 5. Just prior to death she became more intensely cyanotic. At post-mortem the heart showed, as was expected, an auricular septal defect of moderate size, extreme right ventricular hypertrophy, and the pulmonary valve as shown in Fig. 5. We have postulated that, with the sudden release of obstruction at the pulmonary valve, the right ventricular chamber decreased in size, and shunting across the auricular septal defect increased. The onset of an acute auricular arrhythmia may also have contributed to the increased right to left shunt which caused increasingly intense cyanosis even though the patient breathed 100 per cent oxygen. Death appeared due to anoxia. One wonders if simultaneous repair of the auricular septal defect should not be carried out in older patients with this anomaly complicating the pulmonary stenosis.



A. *B.*

Fig. 6.—Patient 41-70-83. Preoperative angiocardiogram showing stenosis of pulmonary artery. *A*, Dye filling out cone of pulmonary valve; *B*, one second later.

The most recent patient to have a pulmonary valvulotomy through a diverticulum sewn to the pulmonary artery probably has an intact auricular septum on the basis of absent cyanosis and an excellent angiocardiogram which was obtained preoperatively (Fig. 6). At operation the stenosis was widely opened and direct catheterization of the pulmonary artery and right ventricle immediately after valvulotomy revealed no gradient between the right ventricle and pulmonary artery to be present (Fig. 7). Although it is realized that

pressures taken at the time of valvulotomy are not entirely significant, the failure to demonstrate a difference between the pressure in the right ventricle and pulmonary artery suggests that the stenosis of the valve has been eliminated.

Aorta.—We have applied the diverticulum to the aorta in one patient. This is the only patient on whom a fabric (Orlon) diverticulum has been used. The aorta was exposed by a third intercostal space transsternal approach.[2] A partially occluding noncrushing clamp[3] was applied to the ascending aorta and an incision exactly 4 cm. in length was made in the pinched-off portion of the aorta. A diverticulum fashioned from Orlon (Fig. 1) was sutured to the edge of the aorta with 4-0 arterial silk. The clamp on the aorta was loosened, but not released, four times at two-minute intervals. When leakage from the fabric ceased, the inside of the diverticulum was carefully flushed out and all clots were removed. The heparin infusion was started into the diverticulum and the finger was inserted in one of the extensions. The clamp on the aorta was

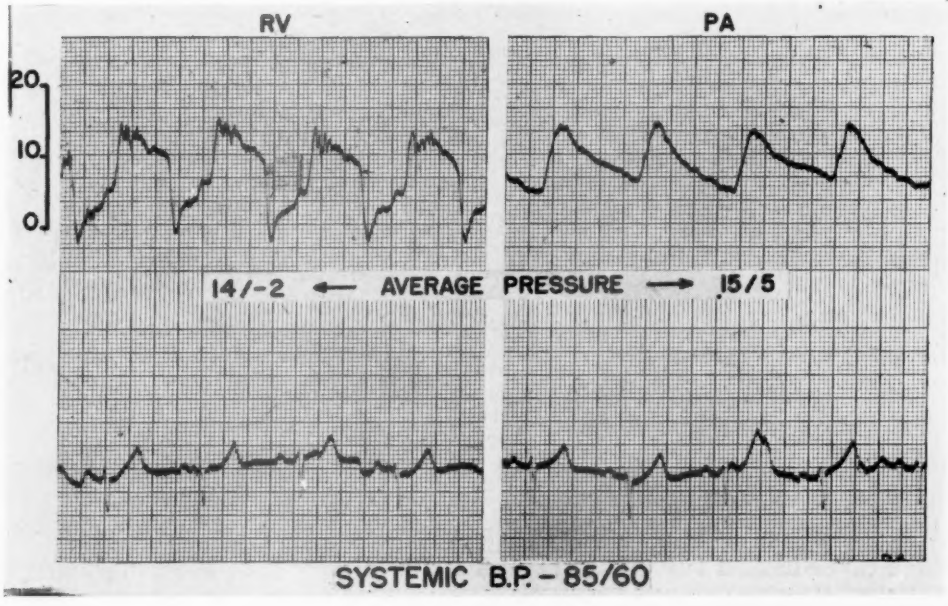


Fig. 7.—Patient 41-70-83. Pulmonary valvulotomy. Pure pulmonic stenosis. Direct pressure measurement following valvulotomy.

then loosened. There was still some oozing from the fabric but this stopped after the aorta was clamped for one additional minute. The valve was palpated, and found to be extremely calcified and distorted. Its lumen would barely admit the tip of the finger. All three commissures could be partially opened and a final opening of at least one and one-half fingers was obtained. There was obviously an enormous increase in flow. The finger was withdrawn into the diverticulum and the aorta reclamped. A second longer clamp was applied below the first, as the aortic wall and incision slipped slightly in the heel of the first clamp. The wound in the artery was closed with a double row of over-and-over 4-0 silk. Several interrupted sutures were also taken. During

the sewing on of the diverticulum, and whenever the aortic clamp was in place, the patient's blood pressure was deliberately lowered with a slow infusion of Arfonad[8] to make the wall of the aorta softer and thicker, and thus less likely to tear in the clamp. This patient survived the operation without any immediate complication but it is too early to determine if this patient will be improved by this procedure.

COMMENT

The approach to intracardiac disease or deformities by the surgeon has been accomplished in three general ways: (1) in the functioning heart; (2) with a temporary mechanical or biologic substitute for the functions of the heart; and (3) in the nonfunctioning heart (venous occlusion and hypothermia). The first is relatively simple but blind and is the one we have been concerned with in this paper. The other two are relatively complex but allow for inspection and repair with visualization of the lesion. Several considerations are important in the determination of the method of approach to be used in any particular case, these include the nature of the lesion, the results of previous surgical and medical therapy with similar lesions, the number of similar cases requiring treatment, the experience of the surgical team, and the availability of an apparatus to aid in the performance of intracardiac surgery. The field of cardiovascular surgery is rapidly expanding and there is ready acceptance by both referring physician and patient of intracardiac surgery. It would seem important to us to promote the development of all of the methods of approach to the heart and great vessels in order that they might be evaluated. It can be expected that there will be improvements in both the simple and the more complex techniques. It is to be hoped that the more simple techniques will prove sufficient for the correction of most, if not all, of the remedial deformities and lesions within the heart. But it is only by the close attention to detail in the performance of the technical procedure and recording of physiologic and pathologic data whenever possible that a proper evaluation of any of the methods of approach to the heart can be made.

The techniques recorded in this report represent our earliest clinical trials with the diverticulum sutured to the functioning heart. It is strongly advised that before they are applied to other patients extensive trial in the animal laboratory be carried out first. It is only in this way that many small points essential to the success of the techniques will become apparent.

SUMMARY

Digital and instrumental surgery in the functioning heart and great vessels has been further explored with the aid of a closed rubber or fabric "diverticulum." Both great arteries and all the chambers of the heart except the left ventricle have been entered through a temporary artificial extension of their lumen. In 12 of 13 cases definitive surgical procedures have been performed within the heart or great vessel. The details of the technique employed and the results obtained are recorded.

We would like to express our great appreciation to Mrs. Joan Hill, Miss Leah White, and Mr. Armand Negri for their technical assistance.

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THE DIAGNOSIS AND TREATMENT OF TRICUSPID ATRESIA

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IN THE past ten years 51 patients with tricuspid atresia have been studied at The Children's Memorial Hospital. Sixteen patients were not operated upon and 15 autopsies were performed in this group. Thirty-five of the patients underwent aortic-pulmonary anastomosis or similar procedures with 10 deaths, of which 7 were autopsied. Observations made upon these patients are the basis of this paper.

PATHOLOGY

The pathology of this condition consists primarily of complete atresia of the tricuspid valve area. Unfortunately, from a therapeutic standpoint, this is not merely a fusion of the valve cusps but an absence of anything simulating valve structure. Solid myocardium separates the right auricle from the right ventricle. There is always an interatrial septal defect. Variations of this basic pattern depend upon the degree of patency of the pulmonary outflow tract and the position of the great vessels.

Four of our patients had Type A variety (Fig. 1) of tricuspid atresia with pulmonary atresia and no interventricular septal defect. The right ventricle was extremely small and nonfunctioning. Blood was able to get to the lungs through a patent ductus in 3 instances and an aortic-pulmonary anastomosis in the other.

Seventeen patients had the Type B variation (Fig. 1) of tricuspid atresia with an interventricular septal defect through which some blood was able to enter the rudimentary right ventricle and pass out into the lungs. In 4 patients there was a normal pulmonary outflow tract without pulmonary stenosis or hypoplasia of the pulmonary artery. In 3 of these the pulmonary valve was bicuspid.

In 3 patients there was pulmonary stenosis, 2 subvalvular and 1 valvular. In the remaining 10 patients of this group there was a moderate to marked hypoplasia of the main pulmonary artery, in 1 instance combined with a bicuspid pulmonary valve.

One of these patients with hypoplasia of the pulmonary artery had a right aortic arch, the only instance in our series of 51 patients. This patient had a ductus arteriosus joining the innominate artery on the left. There were also a sirenomelus and multiple severe gastrointestinal and genitourinary anomalies causing death at 4 days of age.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

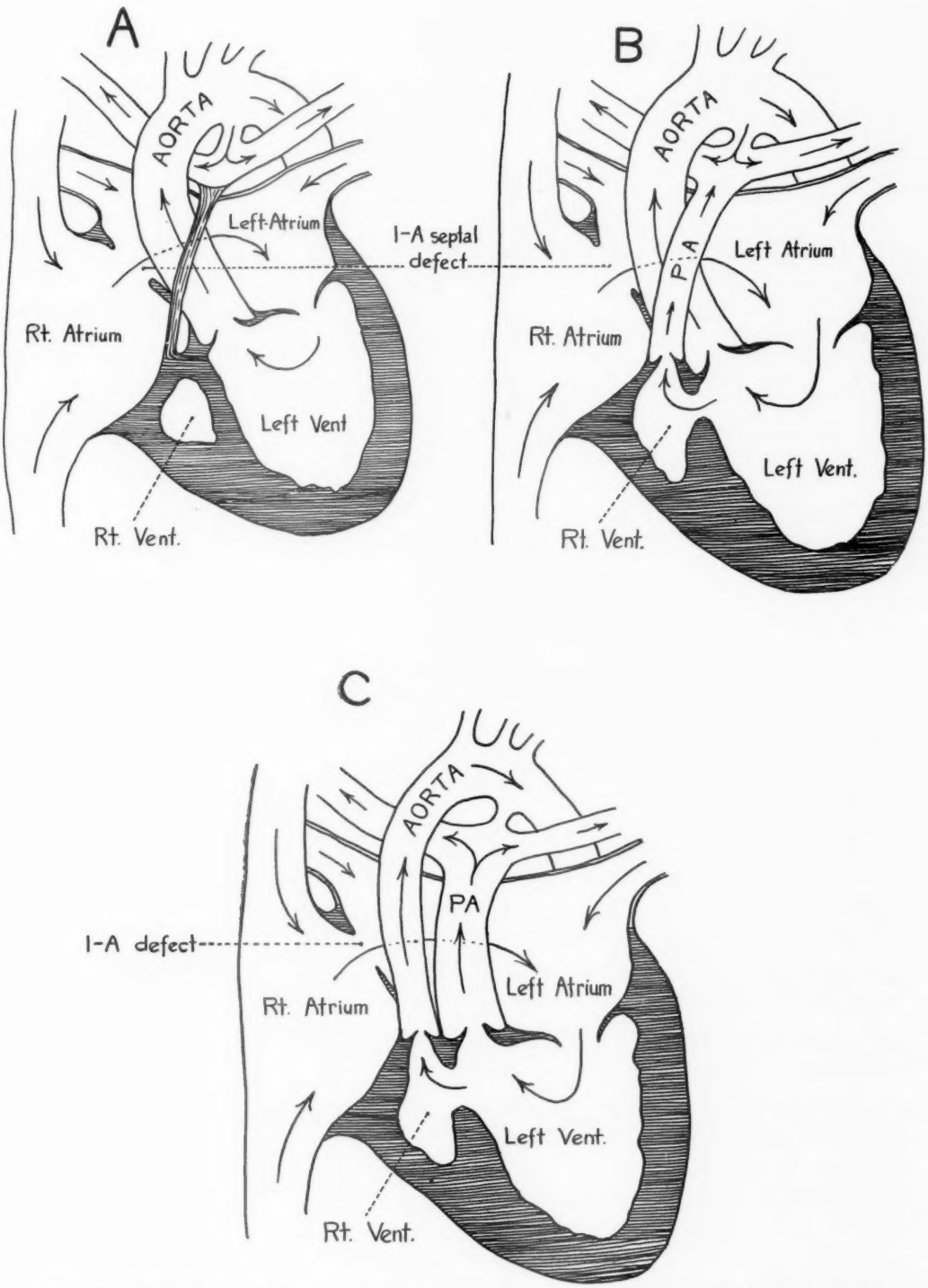

Fig. 1.—Pathologic variations of tricuspid atresia. *A*, Pulmonary atresia without interventricular septal defect; *B*, interventricular septal defect, rudimentary right ventricle, and patent pulmonary outflow tract; *C*, transposition of the great vessels and interventricular septal defect.

The remaining patient had the Type C variety (Fig. 1) of tricuspid atresia with a transposition of the great vessels. The pulmonary artery originated from the left ventricle and the aorta came off the rudimentary right ventricle getting its blood through the interventricular septal defect.

In our case there was no pulmonary stenosis and, in effect, there was a single ventricle. At the age of 3 the child had an exploratory thoracotomy. The pulmonary artery was large and thick-walled with a relatively high pressure (25 mm. Hg). No corrective procedure seemed advisable. The patient did well for five years postoperatively until her death from a brain abscess in 1953.

Another variant of the Type C tricuspid atresia has been described as having a pulmonary stenosis in addition to the transposition of the great vessels.[1] We encountered no example of this in our autopsy material. The most recent of our operative cases was a 9-year-old girl who was thought to have such a combination of defects. An aortic-pulmonary anastomosis was performed because there seemed to be decreased pulmonary flow.

DIAGNOSIS

The presence of left heart strain and peaked, prominent P waves in the electrocardiogram of a cyanotic infant with relatively normal heart size has almost invariably been diagnostic of tricuspid atresia in our series.

A history of cyanosis from birth was almost constant, although in many cases the cyanosis was slight at first, becoming intense during the first few months of life. Dyspnea, weakness, and failure to gain weight were common. In some cases brief episodes of unconsciousness, usually following crying or feeding, were reported. Decreased exercise tolerance was usual in the older children.

Squatting to rest was noted in only 10 per cent of our cases, but the majority had never walked.

On physical examination the cyanosis was usually moderately severe.

Clubbing of the fingers and toes was only slight, if present at all, again indicating the relative youth of these patients.

A loud, harsh systolic murmur was usually heard best in the third intercostal space to the left of the sternum. This was usually described as Grade III to Grade IV intensity. In 6 patients the systolic murmur was short and soft, and in 4 patients no murmur could be heard. There was no definite correlation between the pathologic findings and the absence of the murmur. A systolic thrill was usually palpable.

Cardiac enlargement was only present in 1 patient with congestive failure who died three days after admission to the hospital. The liver was noted to be enlarged in 60 per cent of the cases but was pulsating in only 10 per cent.

Laboratory findings usually revealed a moderate polycythemia averaging 20 Gm. of hemoglobin and 7,300,000 red blood cells.

ROENTGENOGRAPHIC FINDINGS

Roentgenologic examinations indicated a normal heart size in 60 per cent of the patients and only slight enlargement in the remainder, except for the 1 child in acute heart failure. The contour of the heart in the posteroanterior view (Fig. 2) followed a typical pattern. The heart was transverse with a prominent, rounded left border and without the concavity in the region of the pulmonary artery segment so characteristic of a tetralogy of Fallot. Extension of the

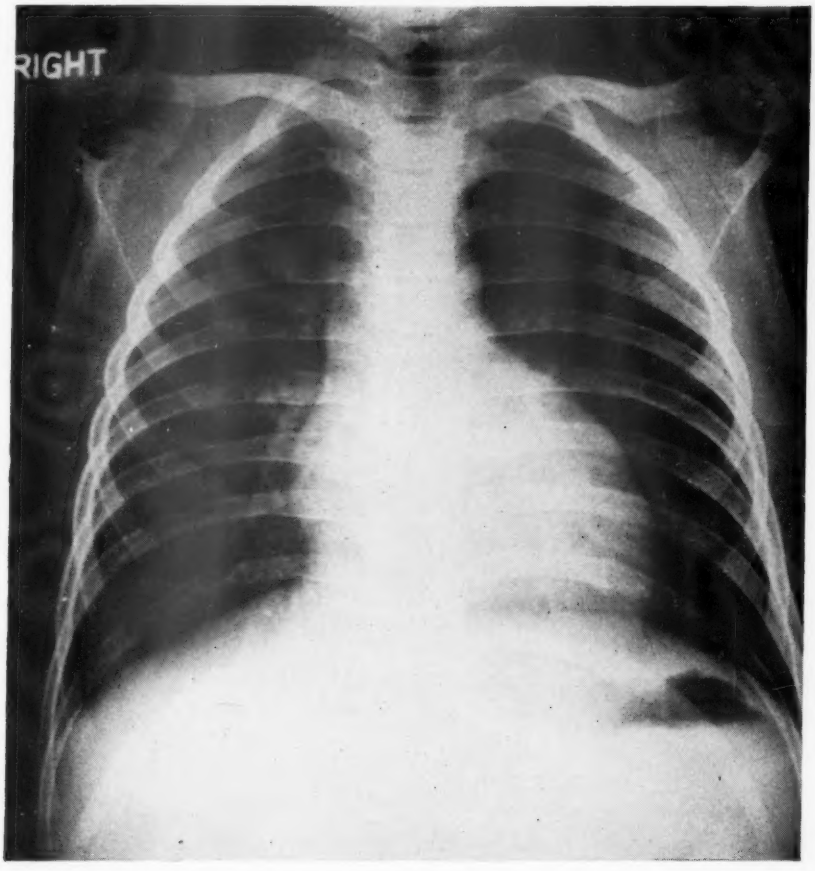

Fig. 2.—Posteroanterior roentgenogram of the chest of a patient with tricuspid atresia. Diminished vascularity of lung fields. Normal-sized, transverse heart with a rounded left border and no concavity in the pulmonary artery segment.

left heart border below the level of the diaphragm was seen infrequently, although this finding has been mentioned as characteristic of tricuspid atresia. There was a decrease in the vascularity of the lung fields in every patient except 1 child who had a transposition of the great vessels with increased vascularity. Oblique views of the chest usually showed a good pulmonary window.

Chest fluoroscopy was helpful in that it indicated absence of right ventricular enlargement as an aid in differentiation from other types of cyanotic congenital heart disease.

The roentgenographic findings in our cases fell into a distinct pattern which served to differentiate them from patients with tetralogy of Fallot.

ELECTROCARDIOGRAPHIC FINDINGS

Evidence of left ventricular hypertrophy was present in every patient in this series. In the infants, of course, this evidence may be no more than the absence of the normal right ventricular dominance. A ventricular complex consisting of a QS, or a small R and deep S wave in the right-sided chest leads was often the only abnormality in the young infants' electrocardiograms (Fig. 3). In the older children, typical left heart strain with peaked P waves in Lead II was practically constant. Tricuspid atresia with right heart strain in the electrocardiogram, occasionally reported in the literature, did not occur in this series (Fig. 4).

Angiocardiogram is seldom necessary unless, in a doubtful case, one wishes to confirm the diagnosis of tricuspid atresia. The characteristic findings are a lack of filling of the right ventricle from the right atrium, a rapid passage of the dye from the right to left atrium and a marked reflux of the dye into the inferior vena cava (Fig. 5). One of our patients died a few hours after an angiocardiogram was made. This might be coincidence; nevertheless, it was disturbing that at post-mortem examination the lesion was found to have been operable.

Catheterization studies are likewise seldom indicated. When the procedure is performed the findings are similar to those of the angiocardiogram, that is, failure of passage of the catheter into the right ventricle and evidence of a large right-to-left shunt at the atrial level.

Since many of these cyanotic infants can tolerate only one major procedure, it usually has been our policy to forego such rigorous diagnostic procedures and operate upon the patients if clinical, roentgenographic, and electrocardiographic findings are typical.

TREATMENT

Eighty per cent of our patients were less than 3 years of age when they came to us for operation or died untreated. Survival into the second decade of life is extremely rare and even those patients are often severely handicapped.

The ideal surgical treatment should always be the return of the organ or system to normal anatomy and function. The realization of this ideal in the case of tricuspid atresia is difficult to imagine in the foreseeable future. The area of the atretic valve would have to be removed and replaced by an artificial valve mechanism that could function efficiently for sixty or seventy years. The septal defects would have to be closed and the pulmonary stenosis or atresia, if present, would have to be corrected. If all of this were possible, we would still be faced with the problem of a rudimentary right ventricle which would suddenly be forced to handle the entire load of the right heart circulation.

At the present time we must be content with making an effort to get more blood to the lungs. All of the systemic venous return passes from the right

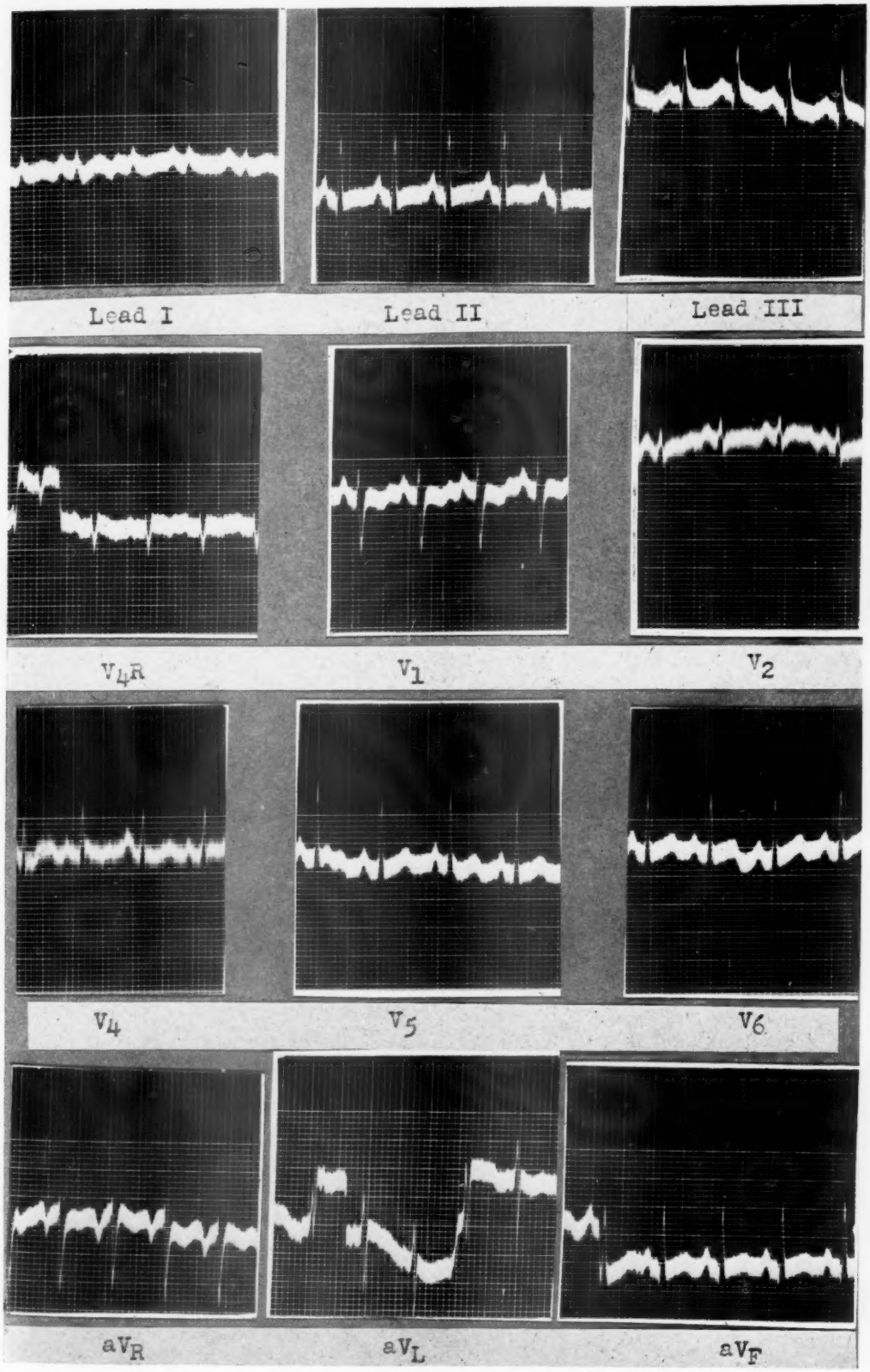

Fig. 3.—Tricuspid atresia in a 2-week-old infant. Note the flat T wave in Lead II and the inverted QRS in V_4R and V_1, normally upright at this age. The P wave is rather prominent in Lead II.

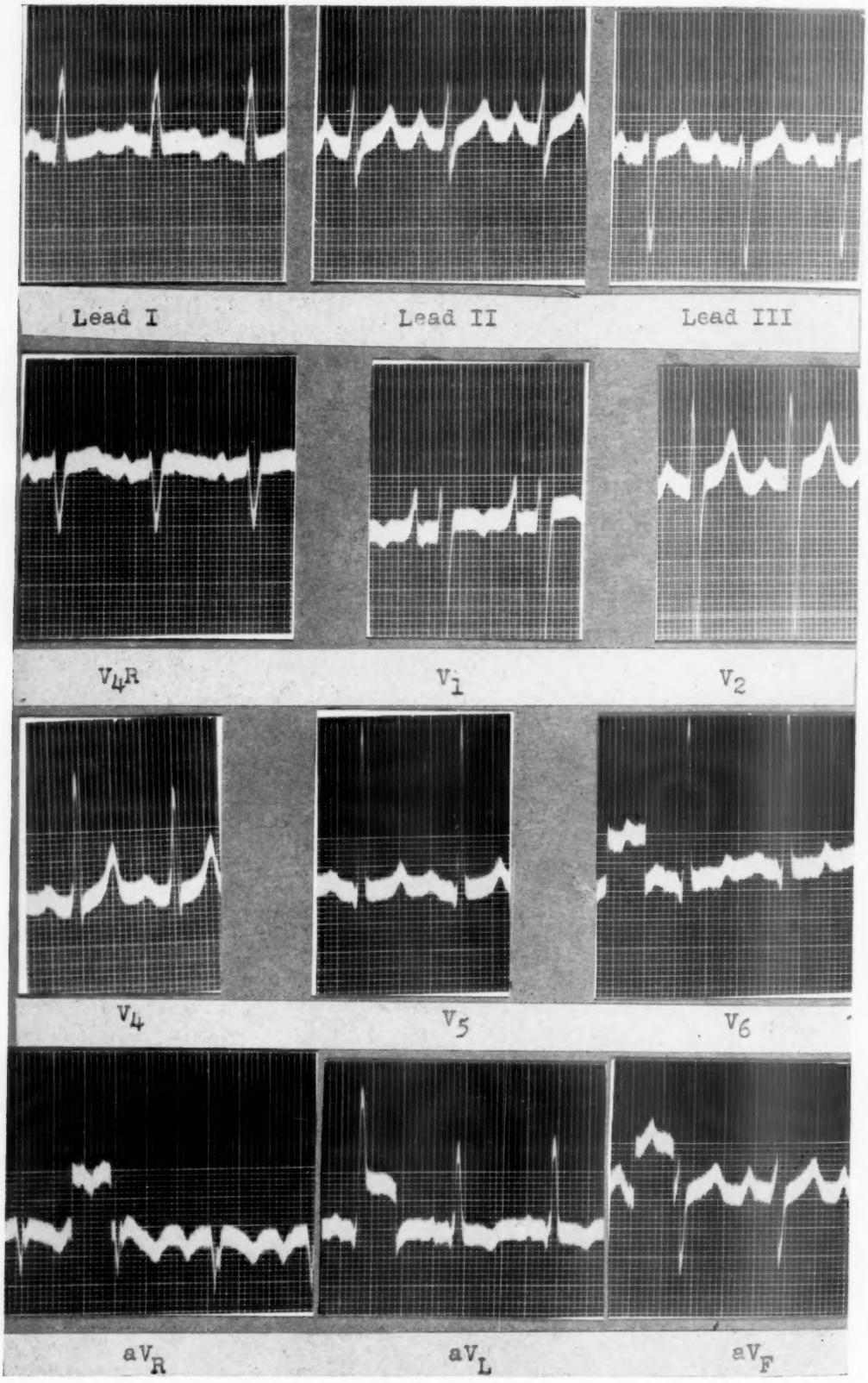

Fig. 4.—Tricuspid atresia in a 7-year-old girl. Note the typical combination of left heart strain and peaked, prominent P waves (Lead II and V_1).

atrium through a large septal defect, usually a widely patent foramen ovale, into the left atrium where there is complete mixing of oxygenated and unoxygenated blood. This mixture, the entire cardiac output, passes through the mitral valve into the enlarged left ventricle. From there it is pumped out the aorta and finds its way to the lungs either through an interventricular septal defect into the rudimentary right ventricle and out into the pulmonary artery or gets to the lungs via a patent ductus arteriosus or through dilated bronchial arteries.

Fig. 5.—Angiocardiogram in a case of tricuspid atresia. Dye does not enter the right ventricle but passes immediately through the interatrial septal defect into the left atrium. There is reflux of dye into the inferior vena cava.

It has been suggested that the interventricular septal defect be enlarged to increase blood flow into the right ventricle and then to the lungs. Even if this were accomplished, the diminutive right ventricular chamber in itself would offer obstruction to blood flow. In addition, 17 of the 21 patients autopsied and

found potentially amenable to surgery had significant obstruction to the pulmonary flow, 4 with complete pulmonary atresia, 3 with valvular or subvalvular stenosis, and 10 with marked hypoplasia of the pulmonary artery. These would offer additional obstruction to the outflow of blood into the lungs.

Since all of the blood flowing into the heart is completely mixed and is pumped out by the effort of the left ventricle, it would seem less difficult and more effective to increase pulmonary blood flow by means of an artificial patent ductus. The efficacy of such an arrangement is well illustrated by the following case report:

C. A. S., a 10-year-old white girl, entered The Children's Memorial Hospital Feb. 14, 1948, with the history of slight cyanosis since birth. She had developed normally and was fairly active, being able to walk at least seven blocks without tiring. For the previous six weeks she had had joint pains and fever. Physical examination revealed 1 plus cyanosis and clubbing. There was a continuous murmur in the second and third left intercostal spaces. The liver was palpable one fingerbreadth below the right costal margin. She had clinical and laboratory evidence of acute rheumatic fever.

In spite of therapy the patient had recurrent attacks of rheumatic fever and died eighteen months later. Autopsy revealed a typical tricuspid atresia and pulmonary atresia with a medium-sized patent ductus arteriosus. The cause of death was a severe rheumatic valvular and myocardial disease. We might presume that an artificial ductus would be equally effective and that had she not developed rheumatic fever we might have expected a much longer survival with minimal disability.

With these facts in mind, on Jan. 6, 1947, the newly devised Potts-Smith operation was performed upon a patient with tricuspid atresia. Since then a total of 35 patients have undergone such anatomoses.

During 1947 and 1948, 13 children were operated upon using the routine method of aortic-pulmonary anastomosis, making the aortic incision $\frac{4}{16}$ to $\frac{5}{16}$ inch long.[2] Five of these patients were over 3 years of age at operation and all had good immediate results. Of the remaining 8 patients operated upon at from 6 months to 2½ years of age, 4 patients died following surgery, 2 of heart failure and 2 due to closure of the anastomosis by thrombosis and pulmonary infarcts. This discouragingly high mortality of 50 per cent in this age group proved the necessity of modifying the usual technique of aortic-pulmonary anastomosis in these small, poor-risk patients.

Gradually, the following operative technique was developed for use in most of the children under 3 years of age, especially for infants only a few weeks or a few months old.

OPERATIVE TECHNIQUE

After the infant's left chest is opened, the pulmonary artery is dissected out as much as possible. It is often only a few millimeters in diameter but has always been adequate for anastomosis in our experience. Forceful traction on the pulmonary artery may occlude the patent ductus, upon which these children are depending, resulting in marked bradycardia and eventually death. Gentleness and constant vigilance are necessary.

The aorta is usually quite small in the patients under 1½ years of age, measuring less than 1 cm. in diameter, but it is quite mobile. Since complete dissection of the

aorta for application of the Potts-Smith clamp is time-consuming and, more important, may involve ligation of important bronchial arteries or occlusion of a patent ductus, only the anteromedial aspect of the aorta is stripped of adventitia in the region adjacent to the left pulmonary artery.

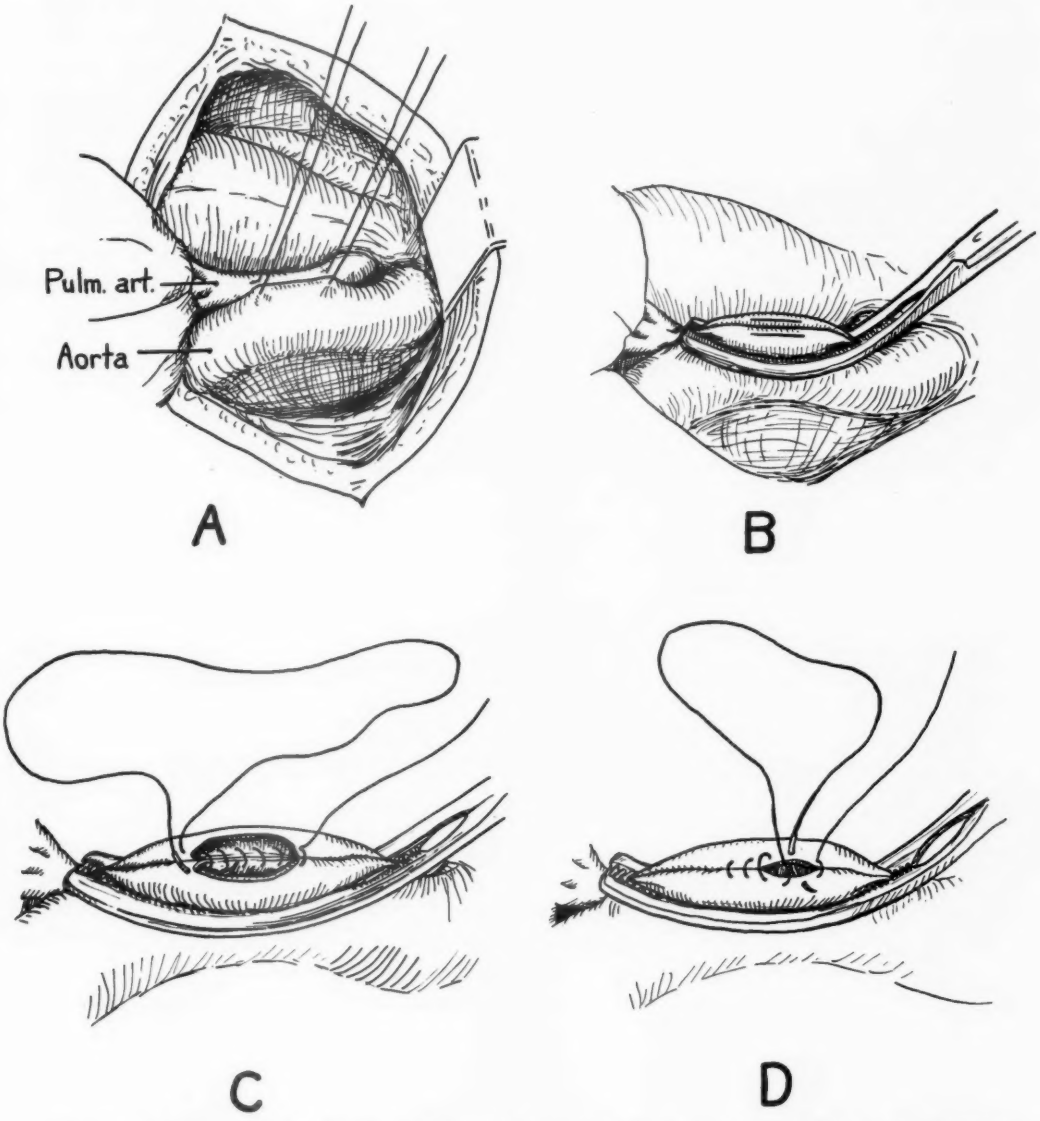

Fig. 6.—An operative technique for aortic-pulmonary anastomosis in infants with tricuspid atresia. *A*, After freeing the pulmonary artery and aorta, two traction stitches are placed 1 cm. apart in the two arteries pulling them up and together. *B*, Curved coarctation clamp has been placed on the two vessels occluding and approximating their lips. *C*, Details of suturing the posterior portion of the anastomosis with a continuous suture of 6-0 silk. *D*, Details of suturing anterior portion of anastomosis.

Two traction stitches of 5-0 silk with swedged-on needle are placed 1 cm. apart, each penetrating all layers of both aorta and pulmonary artery. As these are drawn up they pull up a "lip" of each artery and approximate them (Fig. 6, *A*). A curved, multitoothed Potts coarctation forceps is then applied to the two vessels approximating the segments of the vessels and isolating them from the blood stream (Fig. 6, *B*). Some blood must be allowed to flow through the unobstructed portion of the aorta, but usually it is necessary to

completely occlude the flow of the left pulmonary artery. Blood flow to the right lung through the patent ductus or main pulmonary trunk has always been adequate for the short length of time required to perform the anastomasis.

Parallel incisions 5 mm. in length are made in each vessel and a side-to-side anastomosis accomplished using a single over-and-over suture of 6-0 silk with a swedged-on fine needle. The combination of fine suture material and accurate placement of the stitches diminishes the possibility of thrombosis. Fig. 6, *C* shows that the suture has been started at the proximal angle and tied and the entire posterior row is snugly in place. With such a short opening only about 4 over-and-over stitches can be placed posteriorly, then the corner is turned by coming out of the exact distal angle of the pulmonary incision and back in at the same point in the aortic angle. The suturing is admittedly not easy, due to the smallness of the structures, close coaptation of the openings, and the nearly terminal gasping movements of the patient. The assistant can help a good heal by gently holding the pulmonary artery incision open and steadying the single clamp holding the two vessels.

Fig. 6, *D* illustrates the technique of suturing the anterior half of the anastomosis. After rounding the corner the first stitch or two is pulled tightly but all the rest are left a little slack so that the lips of the vessels can be parted slightly for better visibility while placing the stitches to prevent catching of the posterior suture line.

After completion of the suture line, the stitches are gently tightened with fine-pointed tissue forceps and the free ends are tied.

The entire anastomosis is often performed in less than five minutes. The rapidity of opening the chest, minimal vessel dissection, and speed of anastomosis are all vital to successful operation in these hypoxic infants. After endotracheal intubation, practically no anesthetic agents are used, the child being carried on pure oxygen with controlled respiration. Moderate hypothermia has been used frequently to bring the rectal temperature down to from 30° to 31° C.

Since 1949, 15 children under 3 years of age have been operated upon using essentially the technique described here without postoperative mortality.

Two other patients, 7 weeks and 3½ months of age, could not survive the usual aortic-pulmonary anastomosis because even the slightest traction on the pulmonary artery occluded the ductus and caused preterminal bradycardia. The left pulmonary artery could be occluded distal to the ductus without causing anoxia.

In the first case the proximal cut end of the left subclavian artery was brought down to the distal cut end of the left pulmonary artery and an end-to-end anastomosis was made (Fig. 7, *A*). The infant had been cooled to 27.7° C. During the next one and one-half hours following surgery, the patient gradually was warmed, but then died, apparently of heart failure.

The second case presented a similar problem. With the previous fatality from a subclavian pulmonary anastomosis in mind, this time the distal cut end of the left pulmonary artery was anastomosed to the side of the descending aorta, utilizing a curved coarctation clamp to partially occlude the aorta (Fig. 7, *B*). After six hours of artificial respiration, because the child made no effective respiratory attempts, the patient made an uneventful recovery and for six months now has done quite well without any cyanosis or any of the spells of unconsciousness that she so frequently had preoperatively.

During the same period since 1949, 5 children over 3 years of age also have been operated upon without postoperative mortality.

The obvious advantages of this described variation of the technique of the aortic-pulmonary anastomosis are reflected in the drastic reduction in postoperative mortality from 50 per cent to 0 in this early and critical age group. The speed of the operation is increased and the likelihood of occlusion of a patent ductus or vital bronchial arteries is decreased. Keeping the incision in the vessels down to 5 mm. results in a final internal

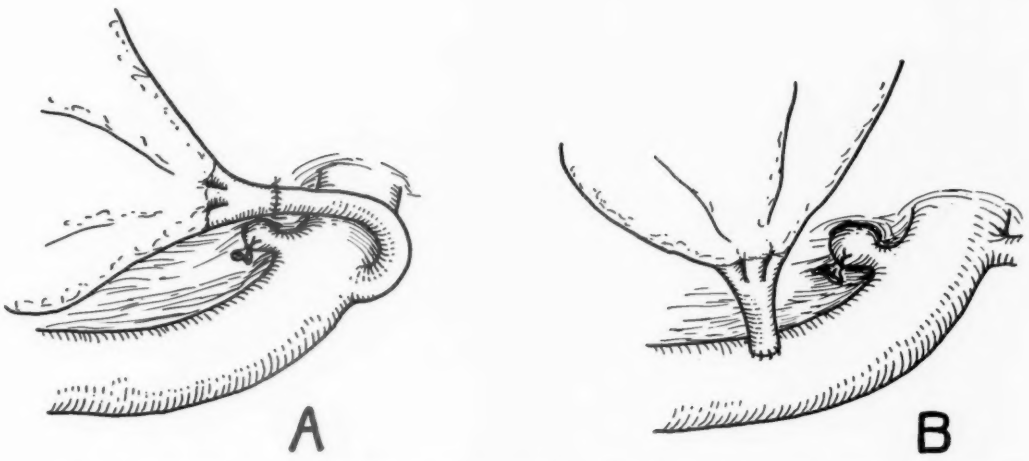

Fig. 7.—Variation of shunting procedures when an aortic-pulmonary anastomosis was impossible. *A*, End-to-end subclavian-pulmonary anastomosis; *B*, end-to-side pulmonary-aortic anastomosis. In each case the right lung is supplied via the patent ductus arteriosus.

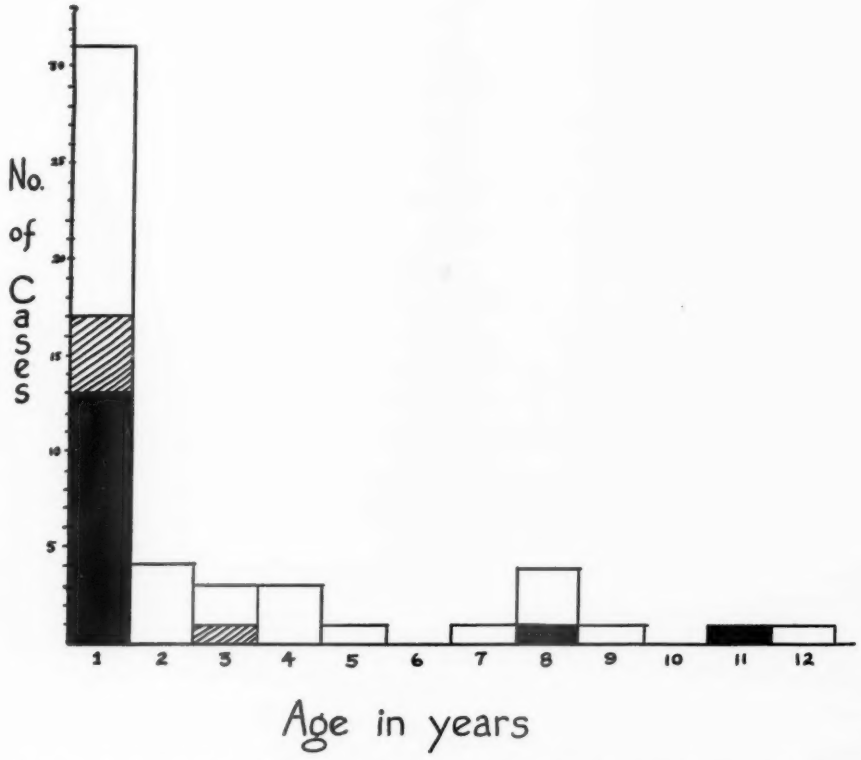

Fig. 8.—Comparison of results and age distribution. Black areas represent patients who have died without operation; shaded areas represent postoperative deaths; light areas represent patients operated upon and surviving; late deaths are not indicated.

diameter of the anastomosis of about 3 mm. (an almost irreducible minimum), and decreases the likelihood of cardiac failure. In order to assure patency the 6-0 suture material and accurate placing of the stitches are essential.

An alternate method of avoiding dissection of the aorta and possible occlusion of a vital ductus arteriosus or bronchial arteries is to apply the curved coarctation forceps to the aorta only. Then the pulmonary artery can be approximated to the aortic lip by drawing distal and proximal traction ligatures on the pulmonary artery across the tip and hinge ends of the blades of the clamp. This technique has been used on several infants with tetralogy of Fallot.

RESULTS

One patient, a 10-month-old boy, was studied in 1948 but was not operated upon. He has not been heard from since then.

Fifteen patients with tricuspid atresia entered the hospital and died without having any type of corrective operation. Thirteen of these patients were less than 6 months of age. Most of them were in poor condition, suffering from spells of unconsciousness and periods of apnea. Many died after only a few days of hospitalization and a few expired while awaiting operation. The other 2 children who died were older and have been reported in some detail previously. Both had defects of a type that allowed adequate pulmonary flow contributing to their survival. One died of a brain abscess and the other of rheumatic fever.

Of the 35 patients who underwent aortic-pulmonary anastomoses, none died during the procedure (Table I). Five patients died from one and one-half hours to fourteen days after operation. Three of these patients died of heart failure and 2 patients died with pulmonary infarcts secondary to thrombosis at the anastomosis.

TABLE I. RESULTS OF OPERATION FOR TRICUSPID ATRESIA (4 MO. TO 8 YR. FOLLOW-UP)

	NUMBER OF CASES	PERCENTAGE
Good results	19	54.3
Fair results	5	14.3
Poor results	1	2.8
Good results but died later	5	14.3
Immediate postoperative deaths	5	14.3
Total	35	100.0

The remaining 30 patients were discharged from the hospital. Two of these patients were considered poor clinical results because of persistent heart failure. The remaining 28 patients were judged good immediate results in that the cyanosis had disappeared, they seemed to be normally active and the spells of unconsciousness, if seen before surgery, did not recur. In a few patients there was evidence of mild congestive failure easily controlled by digitalis.

There have been 5 late deaths in this group. Two were attributable to a combination of the disease and the operation. One 7½-year-old girl died in congestive failure one month after returning home. The other, a 25-month-old boy, died of congestive failure four months after discharge from the hospital.

Two deaths were not related to the disease or operation. One was a 1½-year-old boy who was killed in an auto accident three months postoperatively. The other patient was 12 days old at operation and did very well for one and one-half years only to die of meningicoccemia. At autopsy the anastomosis was patent with a 2.5 mm. internal diameter. The fifth child was 13 months old at operation. He did quite well after operation until five years later when he died of a pneumococcus septicemia following extraction of abscessed teeth. At autopsy there were fresh vegetations at the edge of the anastomosis on the pulmonary side. The anastomosis was patent and measured 3 mm. in internal diameter. This complication could have occurred in any child with congenital heart disease.

The remaining 25 children have been observed for from four months to eight years following surgery. Nineteen of the patients may be classified as good results. They have no cyanosis or only a faint trace on exertion. Their activity is normal although it is always suggested that extremes of exercise be avoided. There is slight increase in the size of the liver postoperatively but no signs of cardiac failure.

Five patients may be classified as having fair results since they have had a mild degree of congestive failure controlled fairly well by digitalis. In spite of this, they are not cyanotic and have good exercise tolerance.

One patient must be classed as a poor result at present. This is the patient with transposition of the great vessels and pulmonary stenosis operated upon four months ago. She is in congestive failure now but it is possible that improvement may occur over the next few months as it has with several of our patients.

Postoperative roentgenographic studies always revealed an increase in the vascularity of the lung fields and some degree of prominence of the pulmonary artery segment. In 10 per cent of the patients there was no cardiac enlargement postoperatively (Fig. 9). In 30 per cent there was slight enlargement and in 60 per cent there was moderate cardiac enlargement. This enlargement mainly involved the left ventricle. Electrocardiograms showed no significant change postoperatively.

DISCUSSION

There seems to be a clinical division of patients with tricuspid atresia according to age groups and symptoms. The children we observed who were operated upon at more than 3 years of age were cyanotic and had decreased exercise tolerance but were never in critical condition. The 10 patients in this age group were operated upon without immediate postoperative mortality. Except for one late death due to heart failure, these patients have done very well over a long-term follow-up period.

The children less than 3 years of age, especially in the first year of life, usually presented a much different picture. These were often infants who became progressively more cyanotic and dyspneic as their ductus arteriosus

narrowed and obliterated. They frequently had spells of dyspnea and deepened cyanosis or apnea, often with loss of consciousness. These attacks were relieved somewhat by oxygen therapy and morphine sulfate. It is this group in our series that has essentially a 100 per cent mortality if no operation is performed.

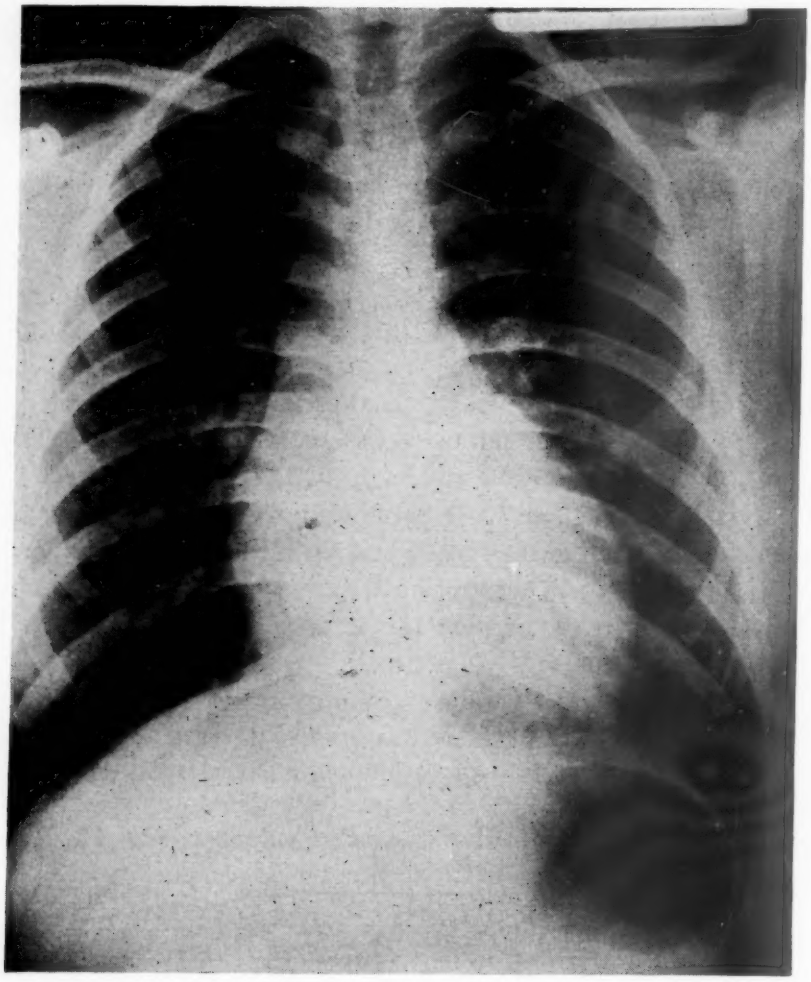

Fig. 9.—Postoperative posteroanterior roentgenogram of the chest of a 15-year-old girl, the same patient as shown preoperatively in Fig. 2. Eight years after operation there is no demonstrable cardiac enlargement, but there is an increase in the vascularity of the lung fields and prominence of the pulmonary artery segment as compared with the preoperative examination.

The high postoperative mortality (20 per cent) in this young age group is not as discouraging as it might seem since the last 16 such patients operated upon with the technique described here have all survived. The results of anastomosis in this age group are most gratifying since an infant with ashen cyanosis in oxygen, having frequent spells of unconsciousness, is converted into a happy, active, and noncyanotic child shortly after surgery.

The good results with operation in this early age group as compared with the nonoperative management would seem to indicate that if an infant with tricuspid atresia becomes progressively more cyanotic and dyspneic and begins to have spells of apnea or unconsciousness, an aortic-pulmonary anastomosis should be performed as soon as possible, even though the patient is extremely young and small.

Fig. 10.—Patient with tricuspid atresia five years postoperatively. Loss of the left leg due to thrombosis of the femoral artery and gangrene preceded the cardiac surgery and might have been avoided if the child had come to operation earlier. She is reaffirming the presence of the continuous murmur of the anastomosis.

Another argument for earlier operation upon these infants with severe symptoms is the frequency of complications. One 10-month-old girl came to the hospital with gangrene of the left leg resulting from thrombosis of the femoral artery. An amputation was necessary and three months later an aortic-pulmonary anastomosis was performed for her tricuspid atresia. Five years later she is in excellent health (Fig. 10). Possibly if the anastomosis had been carried out earlier, this vascular complication would not have oc-

curred. Two other children had cerebral vascular accidents before surgery. One has residual paralysis and remains an invalid even though the cardiac situation is much improved.

Certainly in the case of a patient with tricuspid atresia who has mild symptoms and is in a static condition, operation can be delayed, if desired, until the patient is 2 or 3 years old and anastomosis is relatively less difficult.

SUMMARY

1. The pathologic and clinical findings in 51 cases of tricuspid atresia are reviewed.

2. Aortic-pulmonary anastomoses were performed on 35 of these patients with 5 immediate postoperative deaths.

3. A modification of the usual technique of aortic-pulmonary anastomosis is described which has markedly reduced the mortality rate.

4. In the case of infants with tricuspid atresia showing increasingly severe symptoms, operation should be advised immediately.

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SURGICAL CORRECTION OF CYANOSIS DUE TO ENTRANCE OF LEFT SUPERIOR VENA CAVA INTO LEFT AURICLE

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IT WOULD be difficult to estimate the true incidence of double superior venae cavae. Prior to 1938, when Gross and Hubbard[1] introduced the modern era of cardiac surgery by curing a child with a patent ductus arteriosus, the detection of intrathoracic systemic venous developmental anomalies was largely confined to autopsy reports. Sanders,[2] in 1946, collected 215 post-mortem cases of bilateral superior venae cavae; his was the seventh case with doubling of the azygos veins as well. The demonstration of double superior venae cavae by angiocardiography was first described by Castellanos and associates[3] in 1944, and Miller and co-workers,[4] in 1955, reported 3 cases detected by cardiac catheterization. The 30 additional cases of Winter[5] and the 46 of Campbell and Deuchar[6] include instances in which radiographic and catheterization findings contributed to the diagnosis. The embryologic basis whereby this anomaly derives from a maldevelopment of the left cardial vein system is adequately reviewed in the articles cited.

The left superior vena cava in most instances drains into the right auricle, by way of the coronary sinus, and under such circumstances is of little surgical importance. Because there is a significant association of this malformation with a wide variety of congenital cardiac lesions, however, cases commenced to appear in the literature as they were encountered in the course of operations undertaken for noncyanotic[8] as well as cyanotic patients.[9-11] Undoubtedly, there have been many unreported cases in which this anomaly has been observed incidentally by surgeons. One of us (E. S. H.) noted such a situation in 1951 while correcting a patent ductus arteriosus in a 36-year-old woman, and recalls a similar observation by Gross in 1946. The course of the left superior vena cava has usually been such as not to constitute an anatomic hazard, so that one could complete the intended procedure without concern as regards the venous anomaly.

There are circumstances in which a left superior vena cava may not be an innocuous lesion. Unfortunately, the relatively infrequent instances of drainage of a left superior vena cava into the left auricle have usually been associated with intracardiac abnormalities of such extreme severity that the venous anomaly has not been considered of major clinical note. In this category are the 10 cases collected from the literature by Winter, as well as the 4 new cases contributed by him[5]; the 5 cases described by Campbell and Deuchar[6]; the 4 probable

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

cases of Young and Griswold[10]; and single cases, such as those mentioned by Gould.[12] These reports include examples of left auricular drainage both of a solitary left superior vena cava, as well as an association of this lesion with a right superior vena cava emptying into the right auricle. The high coincidence of absence of the inferior vena cava with the superior caval anomalies has also been noted frequently.[13, 14] Even when associated with a severe tetralogy of Fallot, however, interrruption of a left superior vena cava entering the left auricle may result in considerable clinical improvement, as reported by Diaz and Anido.[9] In their case, the hematocrit of a 7-year-old girl fell from a preoperative level of 91 per cent to 56 per cent postoperatively, with a decrease in cyanosis and an increase in exercise capacity, although no systemic-pulmonary arterial shunt had been constructed. Feindt and Hauch[11] have also described an 8-year-old girl with Eisenmenger's complex, in whom lessening of cyanosis followed ligation of a left superior vene cava as it drained into the left auricle, with a rise in arterial oxygen saturation from a preoperative level of 70 per cent to 84 per cent six weeks later.

The possibility of finding cases of a left superior vena cava emptying into the left auricle in which this constituted the major or only cause for cyanosis has been anticipated. Undocumented statements to the effect that such cases have been encountered may be found,[15-17] although most textbooks pertaining to cardiology either ignore the possibility entirely or dismiss it with a brief negative statement indicating that anomalies of drainage of the venae cavae do not constitute isolated malformations.[18] However, Potter,[19] has contributed a significant case in the autopsy findings of a cyanotic infant with a vascular anomaly of the cerebral meninges who died at the age of 32 hours. This child had an essentially normal heart, with the exception of bilateral superior venae cavae, of which the left superior vena cava emptied into the left auricle; the membrane covering the foramen ovale was described as somewhat unusual. Another pertinent report is that of Gardner and Cole,[20] concerning a woman with severe cyanosis and clubbing, and no cardiac murmurs, who died suddenly at 32 years of age. At autopsy the cause for the cyanosis, which had been noted since the age of 4, was confined to drainage of a large inferior vena cava into the left auricle. The superior caval system was normal, and there were no septal defects. These authors stated that occasionally the left superior vena cava and inferior vena cava have been found to drain together into the left auricle, but that interatrial septal defects have usually also been present.

Cases such as these provide an obvious challenge to the surgeon. The diversion of a considerable volume of venous blood away from the left auricle and back to the right side of the heart can be of considerable benefit. The state of the vascular communications between the right and left caval systems will play a large role in determining the type of surgical procedure to be employed. When, as in the present instance, adequate communicating veins are present, simple ligation or division of the left superior vena cava near its point of insertion into the left auricle may suffice. In the absence of such an extracardiac shunt, transposing the left cava to the right auricular appendage, either by direct-

suture anastomosis or by the use of a graft, would merit serious consideration. An analysis of the patterns of communicating veins may be found in some of the articles cited,[5, 6] as well as in the report of McCotter.[21]

CASE REPORT

T. O., a white girl aged 7 years and 11 months, entered the hospital on Nov. 22, 1954, for evaluation of congenital heart disease. Cyanosis had been noted for the first time at 3 months of age. There had been a slow weight gain during childhood, with continuing cyanosis that increased on crying, dyspnea on moderate exertion, and spontaneous squatting. No evidence of congestive heart failure had been seen. On examination she was an alert,

Fig. 1.—Angiocardiogram from right arm. Right superior vena cava enters right auricle; transverse mediastinal communicating vein visualized.

cooperative child with marked cyanosis of the lips, fingers, and toes, and definite clubbing of the nailbeds in both upper and lower extremities. Her blood pressure was 90/50 in the arms and 120/70 in the legs. There was no venous distention in the neck. The lung fields were clear. The heart was not enlarged, with the point of maximal impulse in the fifth left intercostal space in the midclavicular line. A faint systolic thrill was present, maximal in the the third left intercostal space parasternally, where a harsh systolic murmur was heard that was transmitted to the second and fourth intercostal spaces to the left of the sternum and to the apex. The pulmonic second sound was softer than the aortic. On fluoroscopy no cardiac chamber enlargement was seen, and the pulmonary vasculature was normal. The electrocardiogram showed a large R wave in Leads V_1 and V_R, with an inverted P wave in Leads II and III and a short P-R interval. The blood count and urinalysis were normal.

Angiocardiograms from both arms demonstrated a small right superior vena cava entering the right auricle, a large left superior vena cava entering the left auricle, and an oblique vein high in the mediastinum connecting the 2 venae cavae (Figs. 1 and 2). There was no simultaneous opacification of the opposite auricle or ventricle on either study, and no evidence on cardiac catheterization from both arms of an interatrial or interventricular septal defect; these data will be documented in greater detail in a separate report.[7] A narrowing was noted at the insertion of the right superior vena cava into the right auricle, and also in the region of the origin of the pulmonary artery. The aorta descended on the left, and there was no transposition of the abdominal viscera. Multiple congenital anomalies of the ribs and thoracic vertebrae were seen.

Fig. 2.—*A* and *B*, Angiocardiogram from left arm. Left superior vena cava enters left auricle; mediastinal communication again seen.

Drainage of the left superior vena cava into the left auricle constituted an obvious cause for the cyanosis. The mediastinal venous communication between the smaller right and larger left superior caval systems might have been functioning as an extracardiac interatrial shunt, increasing the amount of nonoxygenated blood being delivered to the left side of the heart. In the opinion of many observers, partial return of the venous blood from the upper part of the body to the left auricle was inadequate by itself to account for the depth of the child's cyanosis. Accordingly, they postulated the possibility of additional anomalous venous drainage from the lower part of the body and/or an intracardiac septal defect. Some degree of pulmonic stenosis was also suggested by the physical signs and angiocardiogram, although confirmatory evidence by catheterization was not available due to inability to pass the catheter through the tricuspid valve. Surgery was elected with the intention of clarifying these problems and of correcting the cyanosis either by obliterating the insertion of the left superior vena cava as it entered the left auricle if the venous communication to the right side were found to be adequate, or by transposing the anomalous vein to the right auricular appendage, if necessary.

Operation was undertaken on Dec. 29, 1954. Under satisfactory general intratracheal anesthesia,* an incision was made across the anterior chest, skirting below both nipples, and

*By Dr. Edith Kepes.

extending from right axilla to left axilla. Both pleural cavities were entered through the third intercostal spaces and the sternum was sectioned with a Gigli saw. The thymus gland, extending onto the pericardium, was mobilized and retracted with traction sutures. The right superior cena cava was smaller than normal, but in the customary location. High in the mediastinum a communicating vein entered the right superior vena cava from the left (Fig. 3). There was no demonstrable azygos system on the right. The pericardium was opened by a vertical incision anterior to the right phrenic nerve, with a horizontal extension over the base of the heart, and the pericardial flaps were held apart with traction sutures. A large vein entered the right auricle in the position of the inferior vena cava. The pulmonary artery was considerably smaller than the aorta, and was directed posteriorly at a more acute angle than normal, at first erroneously suggesting the possibility of a transposition (Fig. 4). A thrill could be felt over the outflow tract of the right ventricle just below the origin of the pulmonary artery, and the pulmonic valve cusps felt normal. The left auricular appendage was small, and the right auricular appendage quite large but not tense. A left superior vena cava, considerably larger than the one on the right, entered the left auricle near the origin of the auricular appendage (Fig. 5). This was joined extrapericardially by a large vein ascending in the left paravertebral groove and presumably representing the azygos system.

The diameter of the right superior vena cava measured 6 mm., left superior vena cava, 16 mm., aorta, 19 mm.; and main pulmonary artery, 12 mm. The mediastinal communicating vein was about the same size as the right superior vena cava, and the hemiazygos approximately as large as the left.

The insertion of the left superior vena cava into the left auricle was mobilized, with considerable caution posteriorly where the vessel was in apposition to the anterior wall of the left pulmonary artery which coursed obliquely behind it. Temporary occlusion of the left superior vena cava resulted in an immediate change in the color of the heart and of the child to a healthy pink, and aortic and left auricular blood samples reflected a normal oxygen saturation (100 per cent plus on the Waters-Conley oximeter with the patient breathing 100 per cent oxygen) in contrast with the cyanotic state, when the peripheral arterial saturation had ranged from 65 to 90 per cent. Following five minutes of left caval occlusion the venous pressure in the left arm had risen only 30 per cent, suggesting that a large quantity of the blood was being shunted to the right side of the heart by way of the mediastinal communication, as well as possibly through other vessels. The demonstration of an adequate by-pass eliminated the need for transposing the left cava to the right auricular appendage. The insertion of the left superior vena cava into the left auricle was permanently obliterated by double ligation with No. 2 white braided silk (Fig. 6). A tube was inserted into each pleural cavity for underwater drainage. The ribs were approximated with pericostal sutures, and the sternum united with periosteal sutures. Running catgut sutures to the thoracic musculature and short continuous sutures of silk to the skin completed the procedure.

Blood samples from both sides of the heart before and after the venous ligation failed to indicate the presence of an interventricular septal defect. Twenty-five minutes after permanent ligation the oxygen saturation of the blood from the right ventricle and main pulmonary artery was 70 per cent. Simultaneous pressure curves, recorded on a multichannel recording apparatus,* revealed a pulmonary arterial pressure of 22-24/10 with a mean of 15, and a right ventricular pressure of 59-63/5, with a mean of 26 mm. Hg. It was felt that the relative pulmonic stenosis was presumably due to the marked hypoplasia of the main pulmonary artery.

The child tolerated the operation extremely well. She exhibited occasional transient episodes of slight cyanosis during the first few postoperative days, but remained pink, thereafter, in gross appearance. The postoperative course was essentially afebrile and uneventful, with primary wound healing, good respiratory excursion, and prompt return to full activity. She celebrated her eighth birthday on the ninth postoperative day. At the time of

*Manufactured by Electronics for Medicine, Inc., New York, N. Y.

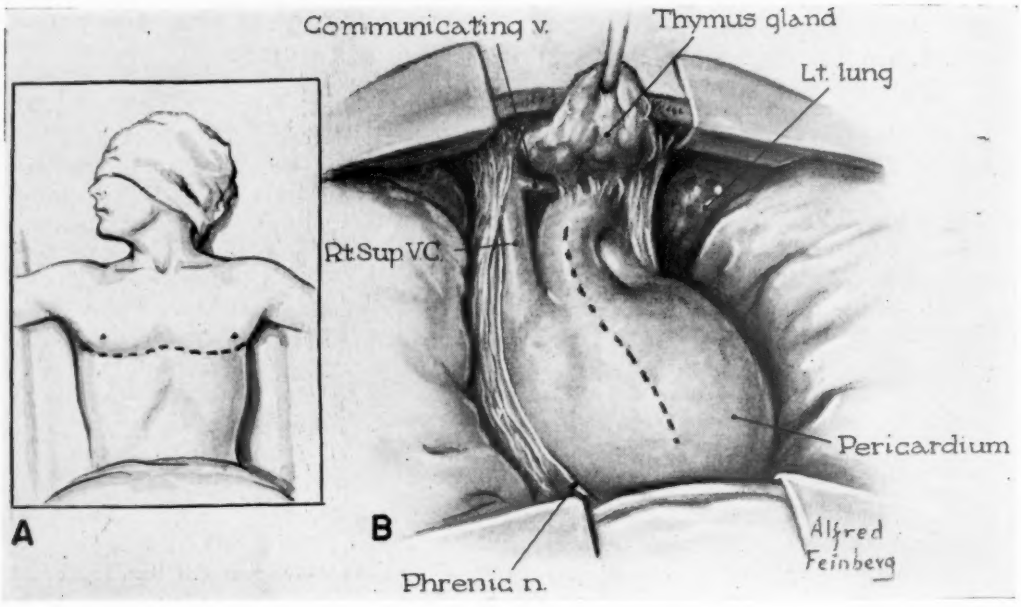

Fig. 3.—*A*, Position and incision; *B*, right superior vena cava and communicating vein, extrapericardially.

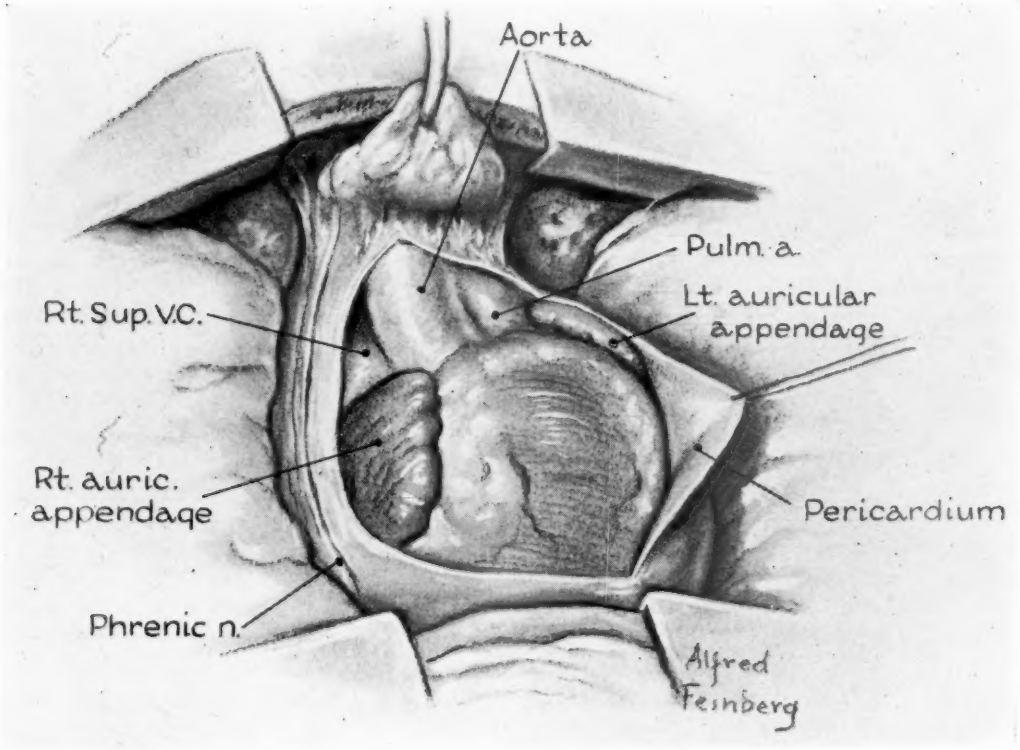

Fig. 4.—Intrapericardial exposure of right superior vena cava and hypoplastic posteriorly placed pulmonary artery.

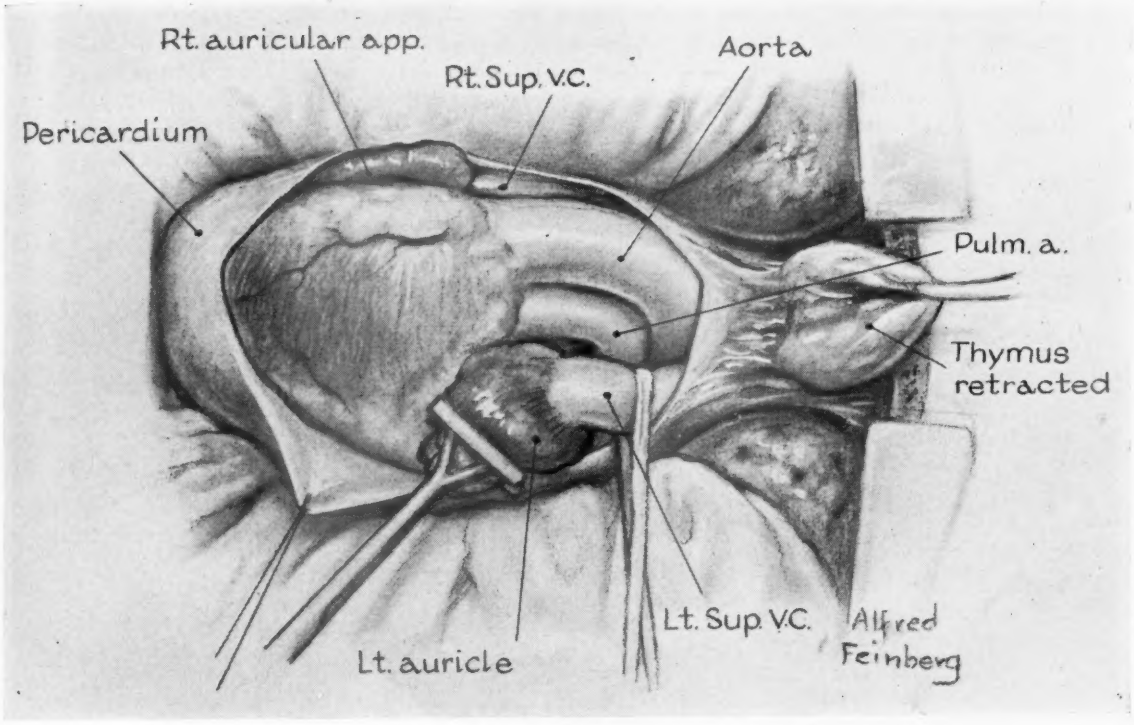

Fig. 5.—Exposure of large left superior vena entering left auricle.

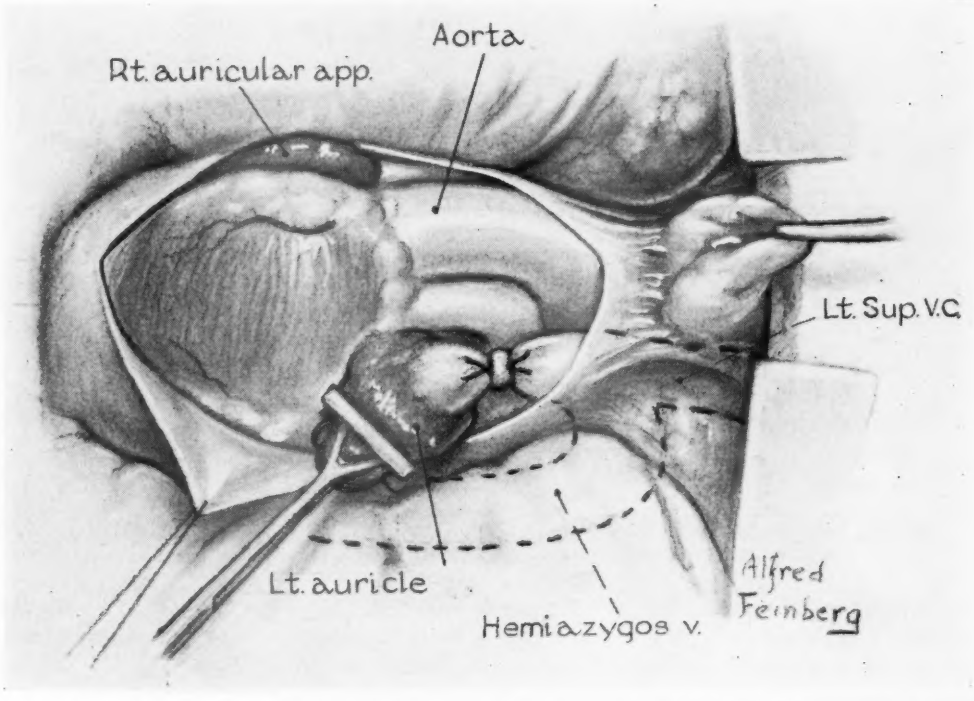

Fig. 6.—Intrapericardial ligation of left superior vena cava. Note large hemiazygos entering left superior vena cava extrapericardially.

discharge, on the twenty-seventh postoperative day, she was still pink. Her venous pressure, however, had remained elevated in the neighborhood of 200 mm. of water, and oximetry indicated a mild degree of residual oxygen unsaturation, correctable by the administration of oxygen.

She was readmitted on March 14, 75 days after operation, for further study of these problems and radiographic demonstration of her inferior vena cava. There had been a

Fig. 7.—Postoperative visualization of inferior vena cava (from left saphenous vein), ascending on left, crossing to right, and entering right auricle; communication with left hemiazygos vein demonstrated in superimposed exposure.

definite increase in appetite and activity, a 5-pound gain in weight, and disappearance of shortness of breath, fatigue, and cyanosis. The child had not squatted since the operation. The superficial veins of the face and upper anterior thorax were slightly more prominent than preoperatively, and the venous pressure was still elevated to about 200. In the absence of congestive heart failure, it was thought that the narrowed entrance of the right superior

vena cava into the right auricle might account for these changes. A suggestive increase in the intensity of the precordial murmur and thrill was attributed to the greater filling of the right side of the heart. The peripheral arterial oxygen saturation was 88 per cent, rising to 99 per cent when oxygen was administered.

Visualization of the inferior vena caval system was accomplished by the injection of 70 per cent Urokon into the left saphenous vein. A large inferior vena cava was seen ascending paravertebrally on the left, crossing the spine obliquely to the right in a caudad direction, then mounting on the right to terminate in the right auricle. At the apex of the

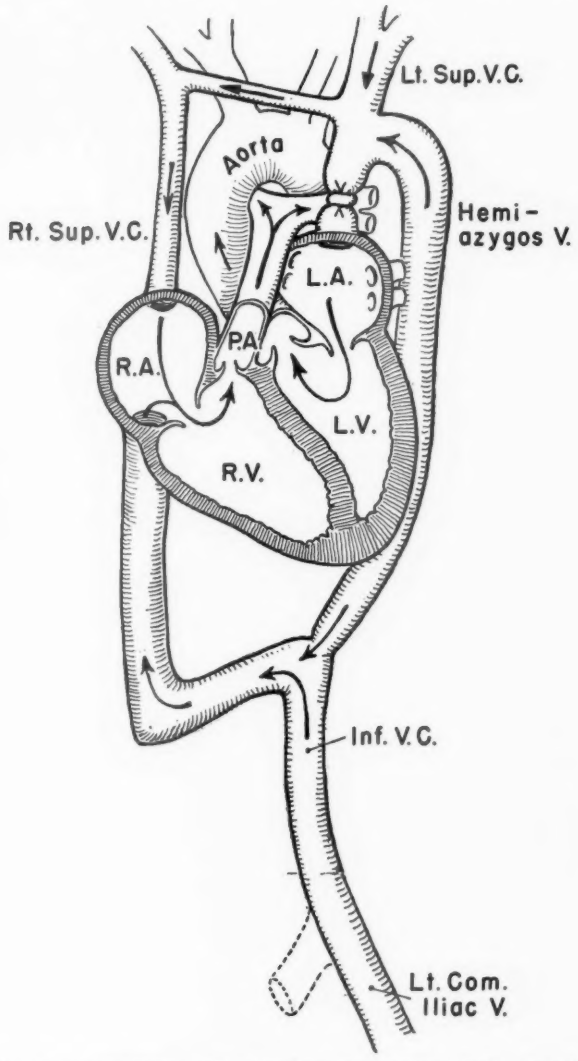

Fig. 8.—Diagrammatic reconstruction of vascular anomalies and ligation of left superior vena cava.

first bend a communication with the left hemiazygos vein was demonstrated, indicating a second major venous pathway for drainage of the left caval system to the right side of the heart (Figs. 7 and 8). The right common iliac vein was not seen, and the possibility of a second inferior vena cava to the right of the lumbar spine was not excluded. No systemic vein was visualized emptying into the left auricle.

At the time of discharge it was obvious that the child had obtained a significant improvement by operation, with disappearance of clinical cyanosis and increase in performance.

Five months postoperatively the clubbing of the fingers had almost completely disappeared, regression of the dilated superficial veins was noted, and the child was able to enter school. The unsolved cause of her residual arterial oxygen unsaturation and her future course in terms of this and the pulmonic hypoplasia will be of tremendous interest in the prolonged follow-up period.

COMMENT

In all likelihood there have been several unreported cases, comparable with the present one, in which surgical attention might well have been directed at the insertion of the left superior vena cava into the left auricle. Gross[22] has encountered such a patient, a young man in whom an erroneous clinical diagnosis of tetralogy of Fallot was made, only to find at autopsy that the major lesion was a left superior vena cava entering the left auricle. The insertion of a left superior vena cava into a normal pulmonary vein should also be amenable to direct surgical correction; this situation was recently found at autopsy in a 2-year-old cyanotic girl at Babies Hospital.[23] McCotter[21] recorded a similar instance, and his bibliography includes references to several remarkable cases from the nineteenth century. He pointed out, as have others,[24] that one or more pulmonary veins may also empty into a left superior cava. Under such circumstances reimplantation of the pulmonary vein(s) into the left auricular appendage would be in order, if the vena cava terminates in the right auricle. Should the cava enter the left auricle, ligation above the anomalous pulmonary vein insertion would effectively correct both abnormalities. McCotter also cited 4 cases in which the coronary sinus drained into the left auricle. This would be clinically unimportant unless associated with a vena cava entering the coronary sinus, in which case correction should be directed at the vena cava. Mankin and Burchell[25] have recorded the case of a 17-year-old noncyanotic girl with an interatrial septal defect, in whom a right pulmonary vein entered a left superior vena cava, which drained into a coronary sinus that emptied into the left auricle. One of the extraordinary cases described by McCotter is that of a 45-year-old man with an otherwise normal heart, in whom a left superior vena cava draining into the right auricle also communicated with the left auricle through a wide foramen measuring 3.5 by 1.5 cm.

The anomalous systemic venous return to the left auricle may consist of a persistent left superior vena cava, an inferior vena cava, or a coronary sinus. In addition, Gould[12] (p. 496) cited 3 examples in which a levoatriocardinal vein arises from the dorsal aspect of the left auricle near the entrance of a pulmonary vein.

The present case cannot properly be regarded as an isolated anomaly, since there is an associated malformation of the pulmonary artery leading to an unusual type of pulmonic stenosis. The residual degree of arterial oxygen unsaturation may also signify a small septal defect that has escaped detection, or additional venous drainage into the left auricle. The remarkable degree of clinical improvement obtained in this case, however, as well as that reported in the 2 cases associated with the tetralogy of Fallot and Eisenmenger's complex,[9, 11] suggests that ligation, division, or transplantation of a left superior vena cava or other anomalous vein entering the left auricle might well be of merit, in

addition to construction of a systemic-pulmonary arterial shunt or direct valvular procedure when these complicated forms of congenital cyanotic heart disease are encountered conjointly. In 1954, Ekstrom[26] performed a Blalock anastomosis plus ligation of an anomalous superior vena cava as it entered the arterial auricle in a 12-year-old girl with tetralogy of Fallot, situs inversus totalis, and double superior venae cavae, each terminating in a separate auricle; striking improvement was noted. An increasing awareness of this lesion as a sole or contributing source of cyanosis should stimulate more widespread utilization of this simple means for diverting venous blood from the arterial circulation.

SUMMARY

1. The clinical significance of vena caval drainage into the left auricle as a cause for cyanosis has been presented.

2. Surgical relief of cyanosis in an 8-year-old girl, due to entrance of a left superior vena cava into the left auricle, was accomplished by intrapericardial ligation of the left superior vena cava.

3. This represents the fourth known case of left superior caval ligation for the relief of cyanosis, and the first in which this constituted the major source of the cyanosis.

4. Whenever a large systemic vein is found to communicate with a left auricle or pulmonary vein, surgical elimination of this cause for cyanosis is recommended, regardless of whether the venous abnormality is an isolated anomaly or associated with intracardiac defects.

Mr. Alfred Feinberg was present for illustrations and Mr. Antol Herskovitz for a motion-picture recording of the operation.

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SYMPATHETIC BLOCK IN APOPLEXY

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APPROXIMATELY 170,000 people in the United States died in 1952 of a stroke and roughly 1,800,000 patients are alive but crippled by this vascular accident.[1] The care of these patients is divided between the general practitioner who sees them first, the neurologist, the neurosurgeon, the internist and institutes of rehabilitation.

Efforts to regard the cerebral circulation as part of the peripheral vascular tree and governed by the same hemodynamic principles has not gained much favor. In a recent paper presented before the American College of Physicians,[2] I summarized the arguments against and for the use of cervical sympathetic block in the immediate treatment of certain forms of apoplexy and they will not be repeated here except to restate that the vasomotor control of the cerebral vessels, which does not serve heat regulation, is admittedly weak and that even short and partial attacks of regional anoxia will readily result in cerebral edema to the detriment of the ganglion cells and the glia. The previous literature was summarized in this article.[2]

Since 1948, when the late Dr. N. C. Gilbert and I[3] first reported a small group of patients treated by early cervical sympathetic block, the vascular surgical service at St. Luke's Hospital has continued to use this method in a few well-selected cases. It is the purpose of this paper to discuss indications, case selection, technique, and results, and to try to draw some tentative conclusions as to the mechanism by which sympathetic interruption may be of benefit.

Indications for Sympathetic Block in Apoplexy.—Strokes occurring in brain tumors, in subdural, subarachnoid, and intracerebral hemorrhage, when recognized, are obviously excluded from this treatment and need immediate neurosurgical consultation.[4] While in a neurological institute such lesions may predominate,[5] certainly the material of a general hospital is different. At St. Luke's Hospital between 1940 and 1946, Gilbert and I studied 121 patients with apoplexy and found 15 emboli, 53 thromboses, and 53 hemorrhages. The diagnostic criteria used in that study,[3] based on neurological interpretation,* have undergone considerable revision since that time in the light of more recent studies.

In the first place, embolism is still the most clear-cut syndrome of the three. Such patients have an obvious cardiac lesion to account for the source of the embolus or may already have had previous embolic episodes to the brain or elsewhere in the body. Their spinal fluids are clear and show no deviation of

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

*Dr. R. P. MacKay, senior neurologist, examined most of these patients.

pressure. Rheumatic hearts with or without fibrillation, a recent myocardial infarct, or a recent commissurotomy make the sequence of events clear. And yet the resulting infarct may present a massive hemorrhage as shown in the brain of patient C. W. (Fig. 1). The spinal fluid of this patient was negative at first and not re-examined until the tenth day, when it was frankly bloody. Such an experience would make one very cautious in advocating anticoagulants for cerebral embolism, especially in the older age group.

Fig. 1.—Brain of C. W., aged 44, who had had repeated admissions to St. Luke's Hospital for rheumatic heart disease, auricular fibrillation, and cerebral embolism from which spastic paralysis resulted. On her last admission she was again regarded as having a cerebral embolus with negative findings in the spinal fluid. Note the massive intracerebral hemorrhage with old areas of encephalomalacia and many punctate hemorrhages. (St. Luke's Hospital Autopsy 41/1941.)

On the cardiovascular service, on a patient who has had one or more peripheral emboli and exhibits a mitral stenosis, mitral commissurotomy is performed instead of administering long-term anticoagulant therapy. Thus far, in about 200 patients operated upon* no further emboli occurred but the duration

*By Dr. Ormand C. Julian and his associates.

of follow-up is relatively short. These data do not include emboli occurring during or within a few hours of commissurotomy. Dr. John Kirklin,[24] in approximately 200 cases of his own, knows of two late emboli. The operation thus is fairly protective for emboli from the left side of the heart and is preferable to long-term anticoagulant therapy.

Fig. 2.—Saccular aneurysm of the basilar artery with large subdural hemorrhage, old myocardial infarct, and bilateral popliteal aneurysms. Onset of symptoms was slow; weakness and tingling with increasing drowsiness preceded the final coma for weeks. (St. Luke's Hospital Autopsy 278/1948.)

The diagnosis of cerebral thrombosis, as hinted in the first paper with Gilbert,[3] rests upon uncertain grounds. Hicks and Warren[6] presented evidence that in 60 of 100 autopsies undertaken in cases of apoplexy, no arterial thrombus or occlusion could be found. Cerebral infarction, then, may occur as a result of cerebral vascular insufficiency analogous to a coronary insufficiency without thrombosis[7] or as a result of cerebral vasospasm, demonstrated in the experiments

of Byrom[8] in hypertensive encephalopathy. That the gradual onset and slowly increasing hemiparesis is not always due to a thrombotic episode is illustrated by the case of R. S. (Fig. 2) whose slowly leaking basilar aneurysm simulated a "cerebral thrombosis."

Even more difficult is the diagnosis of cerebral hemorrhage. Obviously, massive blood in the spinal fluid with fulminating onset in a hypertensive patient, provides the diagnosis of intracerebral hemorrhage. I am not discussing here, of course, cases of subarachnoid hemorrhage. But 30 per cent of our

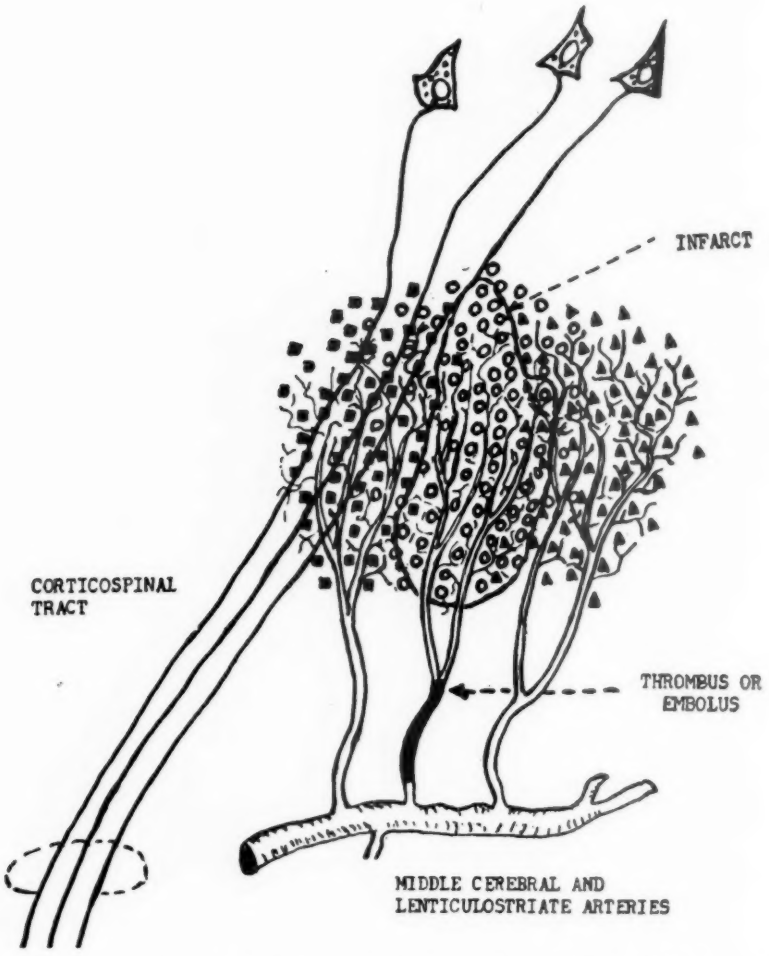


Fig. 3.—A lenticulostriate artery has been occluded. The completely encircled zone undergoes complete necrosis. Most of the parenchymal and interstitial cells are destroyed. In the intermediate zone around the ischemic center only the more vulnerable nerve elements die, leaving the supporting tissue intact. Circles, triangles, and squares overlap. The corticospinal tract traverses the periphery of the ischemic zone, where temporary interruption of function occurs without any visible alteration of structure.[9]

intracerebral hemorrhages had a clear spinal fluid, even though a second lumbar puncture taken four to five days after the acute onset may have revealed bloody or xanthochromic fluid. There seems to be no evidence that if cervical sympathetic block is performed in a case of unsuspected hemorrhage, bleeding would

increase. This, however, cannot be said of anticoagulants and one marvels at the temerity of those who advocate long-term anticoagulant therapy in cases of cerebral embolism and thrombosis,[1] the diagnosis being as uncertain as it is. It has been our practice, then, that if intracerebral or extracerebral hemorrhage and brain tumor can be reasonably excluded, cervical sympathetic blocks are carried out first on the side and a few hours later contralateral to the cerebral lesion.

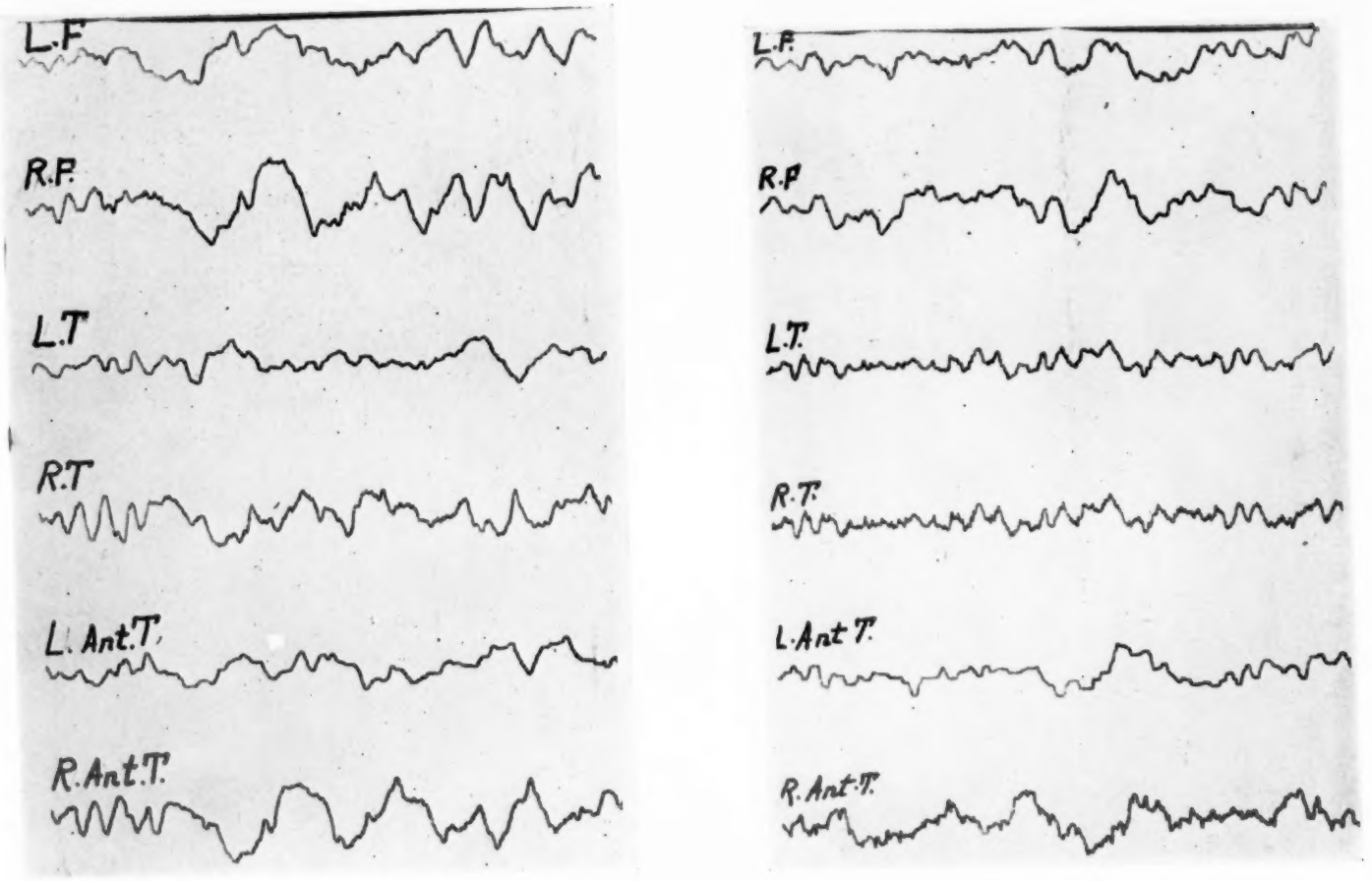
Regression of Widespread Neurological Symptoms in Cerebral Vascular Accidents.—The fact that a massive flaccid paraplegia or hemiplegia frequently terminates in limited residual damage clearly indicates that focal cerebral ischemia leading to necrosis must be surrounded by a type of cerebral lesion which is reversible. Fig. 3* clearly depicts the ischemic effects of occlusion of a lenticulostriate artery.[9] The completely circled zone is necrotic; both the parenchymal and interstitial tissues are destroyed. In an intermediate zone around the focal necrosis only the nerve cells and the more vulnerable parenchymal elements are destroyed, but the supporting tissue is intact. In the most peripheral part of the ischemic focus, the edema and stasis derange function, but no permanent alteration in structure takes place. Thus, the corticospinal tract, which traverses the periphery of the cerebral infarct, begins to function as soon as the perifocal edema subsides.

This concept was clearly enunciated by Leriche[10] at the time that he blocked the sympathetics in two postoperative hemiplegics. He spoke about a halo of edema around the ischemic focus, but stressed vasospastic phenomena. It is not necessary, however, to assume any release of vasospasm in explaining the sudden improvement in some hemiplegics after sympathetic paralysis. My co-workers and I have repeatedly emphasized that in segmental occlusions of peripheral arteries the release of vasomotor tone decreases peripheral resistance, increases collateral flow, and thus improves tissue nutrition without having to invoke the elimination of vasospasm.[11] Edema due to anoxia or hypoxia of the capillaries occurs very readily in the brain and is seen together with punctate hemorrhages around fresh cerebral infarcts.[9]

Electroencephalograms, of which an example is shown in Fig. 4, reveal the greatly improved appearance of slow-wave foci, in this instance in the right temporal, right anterior temporal, and bifrontal leads. The change in the record occurred within fifteen minutes of the injection of procaine.

Material Studied.—Of 83 apoplectic patients, 55 received injections to the cervical sympathetic trunk. Of the 55 injected patients, emboli were diagnosed in 14, cerebral infarct in 41, 23 had massive intracerebral hemorrhage, 1 a subarachnoid hemorrhage, 1 a cerebral venous thrombosis, and 3 had brain tumors (Table I). There is no evidence that harm would have come to the noninjected group after injection, and, as a matter of fact, patients with either intracerebral hemorrhage or brain tumor have been inadvertently injected, with temporary improvement, indicating the existence of edema around the lesions; nevertheless, we have not deliberately injected them, nor were cases of brain concussion treated[12] since we have no access to such material. Thirty of the 55 patients received only one injection, whereas the rest of them received from two to six injections (Table II). Plastic catheters were

*Diagram by courtesy of Raymond D. Adams.



A. *B.*

Fig. 4*A*.—Electroencephalogram (before nerve block) of a 76-year-old man admitted to St. Luke's Hospital on May 27, 1948. The record was taken 6 hours after a gradual onset of a left-sided hemiplegia and shows an irregular slow-wave focus of 1½ to 2 seconds' frequency in the right temporal, right anterior temporal, and bifrontal leads. Background activity about 7 seconds. Impression: slow-wave focus centering in the right frontotemporal area.

Fig. 4*B*.—Tracing obtained on the same patient 15 minutes after a right cervical sympathetic block productive of a Horner's syndrome. All slow activity decreased. Focus reduced and entire record looks normal. Impression: great improvement. (Records reported by Frederick J. Gibbs.)

inserted in 2 patients for five and seven days, respectively, and bilateral injections were carried out on 5. In a more recent group, not included here, all patients received bilateral injections six hours apart for reasons which will be discussed.

The influence of the time factor is shown in Table III. Contrary to our expectations and to our early experience, the 20 patients treated between twenty-four and forty-eight hours still showed improvement in a little over half of the cases and there are some favorable responses reported and unreported several days after the vascular accident.

TABLE I. SYMPATHETIC BLOCK IN APOPLEXY

DIAGNOSIS	NO. OF CASES	IMPROVEMENT	FAILURE
A. Injected			
Embolus	14	14	—
Infarct	41	18	23
Total injected	55	32	23
B. Not injected			
Massive hemorrhage			23
Subarachnoid hemorrhage			1
Cerebral venous thrombosis			1
Brain tumor			3

Among the late sequelae of cerebral vascular accidents, a causalgic type of pain occurs rather infrequently in the hemiplegic extremity. When it does occur, this thalamic pain is highly resistant to treatment. In a previous report on causalgic states this lesion of the third sensory neuron was described.[13]

TABLE II. NUMBER OF INJECTIONS RECEIVED BY APOPLETIC PATIENTS

NUMBER OF PATIENTS	NUMBER OF INJECTIONS
30	One (30)
10	Two (20)
9	Three (27)
4	Five (20)
2	Six (12)
Total 55	109

Two patients had an indwelling plastic catheter for 5 to 7 days, respectively; 5 patients received bilateral sympathetic blocks.

TABLE III. TIME FACTOR IN THE TREATMENT OF APOPLEXY

TIME ELAPSED SINCE ONSET (HOURS)	NUMBER OF CASES	IMPROVEMENT	FAILURE
3-6	15	10	5
6-12	10	7	3
12-24	5	3	2
24-48	20	11	9
More than 48	6	1	5
Total	56	32	24

In a patient studied in 1949, who exhibited a frozen shoulder and a painful, swollen left upper extremity, left-sided sympathetic blocks seemed of no avail. The left hemiplegia was the result of a right carotid angiogram performed in another institution. Great relief and almost complete restoration of function resulted from two cervical sympathetic blocks on the right, on the side of the infarction. This experience would indicate that while motor improvement can hardly be expected at a late date, sensory phenomena are still capable of improvement. The injections were helpful not because of their peripheral vasodilator or pain-blocking effect, but on the contralateral side, on the side of the infarct.

While there can be no set time limit for the efficacy of injections, a sudden improvement after block can hardly be expected after forty-eight hours.*

Technique of Injections.—The lateral approach at the level of the sixth cervical transverse process is used, as a rule (Fig. 5), but the anterior approach has been favored by many of our residents and also most of the anesthesiologists. These methods have been carefully described and illustrated in detail by Moore[14] and need not be discussed here. If a Horner's syndrome does not appear within fifteen minutes, the block has to be repeated; the opposite chain is not injected for four to six hours. Ten cubic centimeters of 1 per cent procaine is used without epinephrine; phenol has never been injected at this level. For the last six years, injecting at the level of the sixth cervical vertebra, no untoward symptoms have been observed, although a conglomeration of surgical, medical, and anesthesia residents have performed well over 200 cervical sympathetic blocks.

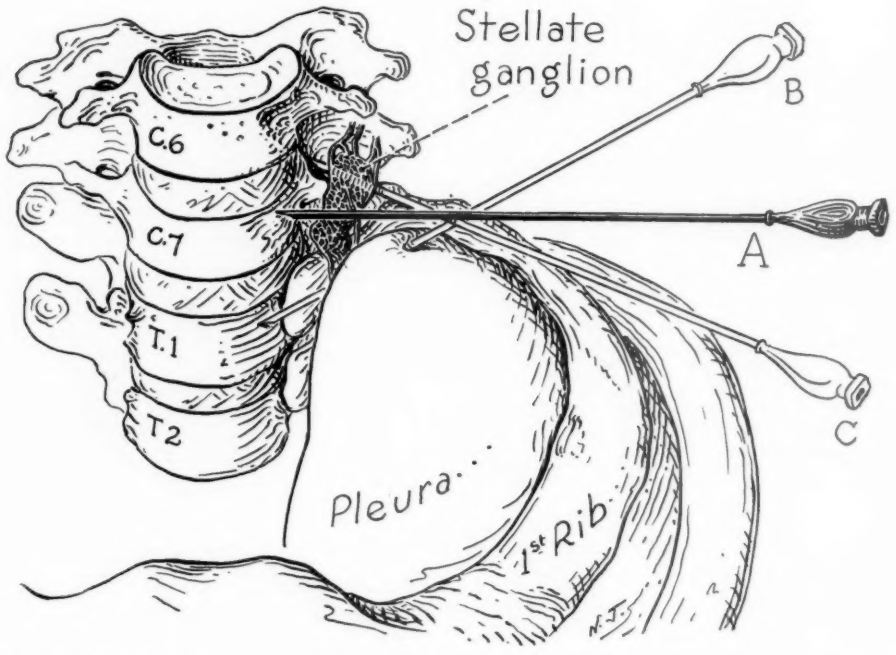


Fig. 5.—Lateral approach to the cervical sympathetic truck. *A*, Correct position of the needle at right angles to the spinal column; *B*, the needle has been directed caudally to establish contact with the first rib which is unnecessary; puncture of the pleura or lung may result; *C*, the needle has been directed cephalad and may pierce the dural sleeve or enter the intervertebral foramen.[3]

RESULTS

In Table IV the degree of improvement following cervical sympathetic block is listed. The results shown occurred within fifteen to thirty minutes following the nerve injection and cannot be attributed to the gradual regression of neurological symptoms. When improvement failed to appear despite a

*This was in the case of an orchestra conductor who, on hearing a record of "Tosca" which he was due to conduct, received a severe setback in the muscular power of his left arm and hand five days after a hemiplegia which was responding well to injections. No further improvement could be obtained after this episode.

definite Horner's syndrome, the injections were not continued, but if improvement was noted the injections were continued twice a day, one injection for each side, until the patient's condition became stationary.

The block failed in 23 of 55 patients, that is, in 41 per cent of the cases. As possible causes of failure the following factors may be cited:

1. *Spreading Intracerebral Hemorrhage and Edema.*—The spinal fluid may not be bloody at the first tap, but blood does appear four to six days later, when the hemorrhagic infarct reaches the cerebrospinal fluid system.

2. *The Time Factor.*—Our best results have certainly been obtained in the early cases and no clear-cut improvement has been seen after forty-eight hours in this series.

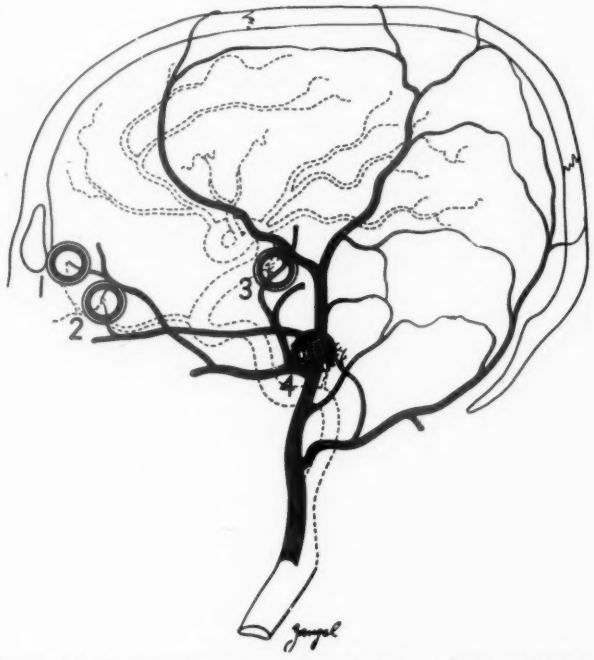


Fig. 6.—Anastomoses between the external and internal carotid arteries. Branches of the external carotid connect through the angular, supraorbital, lacrimal, and nasociliary artries with branches of the ophthalmic. *1*, Anterior meningeal with posterior meningeal; *2*, ophthalmic with internal and external maxillary; *3*, lacrimal and nasociliary with internal maxillary and occipital; *4*, caroticotympanic with internal maxillary arteries.[2]

3. *The Availability of Collateral Blood Supply.*—The area of perifocal edema may be vascularized through a group of channels which include the circle of Willis, the homolateral external carotid, and the meningeal arterial anastomoses. In a case of cerebral arteriolar sclerosis, even the most potent vasodi-

TABLE IV. IMPROVEMENT FOLLOWING CERVICAL SYMPATHETIC BLOCK (55 PATIENTS)

	PATIENTS	PER CENT
Consciousness regained	10	17
Speech regained	7	12
Speech improved	15	27
Motor improvement	12	21
Flaccid paralysis abolished	8	14
No improvement	23	41

lator, CO_2, is unable to effect an increase in blood flow[15] and sympathetic block cannot improve circulation. This is known to be the case in arteriolar sclerosis of fingers and toes.

None of these factors relating to available collateral circulation is known at the time of the vascular accident. The circle of Willis is variably developed and under normal conditions the blood of the two internal carotids does not mix. Only when a vascular block develops will the communicating arteries of the circle of Willis come into play. The normal pattern was present only in 50 per cent of the cadavers.[16] Thus, the anomalies of the circle of Willis have a decisive influence upon the development of cerebral ischemia.[17] The communication between the internal and external carotid arteries can be visualized and a

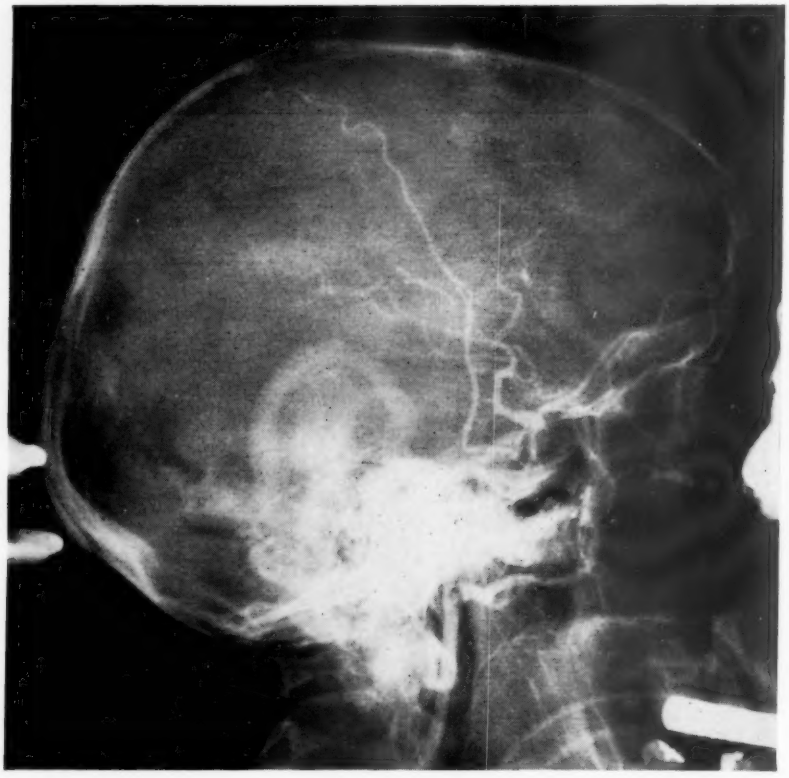


Fig. 7.—This is one of six arteriograms taken within five seconds. In none did the internal carotid artery fill. The external carotid with its external maxillary branch can be seen. The middle cerebral artery and superficial temporal arteries are also filled. The middle cerebral artery thus was filled through an anastomosis through the external carotid artery, probably the ophthalmic. (Courtesy of Dr. A. VerBrugghen.)

diagram illustrates the possible connections (Fig. 6). In a cerebral angiogram, taken in a patient suffering from a thrombosis of the internal carotid artery, one sees a definite filling of the middle cerebral artery through the homolateral external carotid artery (Fig. 7). In addition, the angiograms of de Sousa-Pereira[18] and others have shown an increased vascular pattern after temporary sympathetic interruption. My neurosurgical colleagues have not felt it advisable to perform angiograms immediately after apoplexy. There is, however, evidence

in the literature that spasm is demonstrable in 90 per cent of the patients after strokes.[19] That emotional stress may bring on apoplexy is known and Ecker has presented interesting case histories of this sort.[20]

The extent of previously occluded arterial pathways producing silent and compensated infarctions is also unknown. When a functioning collateral serving the area of a previously blocked vessel is suddenly occluded, a vast area of ischemia develops and the block cannot be of much avail. Patients with second or third strokes have not responded as well to blocks as after the first one.

For the reasons cited, one can never be sure at the time of apoplexy how much of the cerebral damage is reversible and here again we find an analogy with an ischemic limb, in which the acute arterial occlusion might be relieved by sympathetic block or embolectomy, but might be unsuccessful because of previous emboli, often unknown to the patient, which block effective collaterals or terminal pathways.

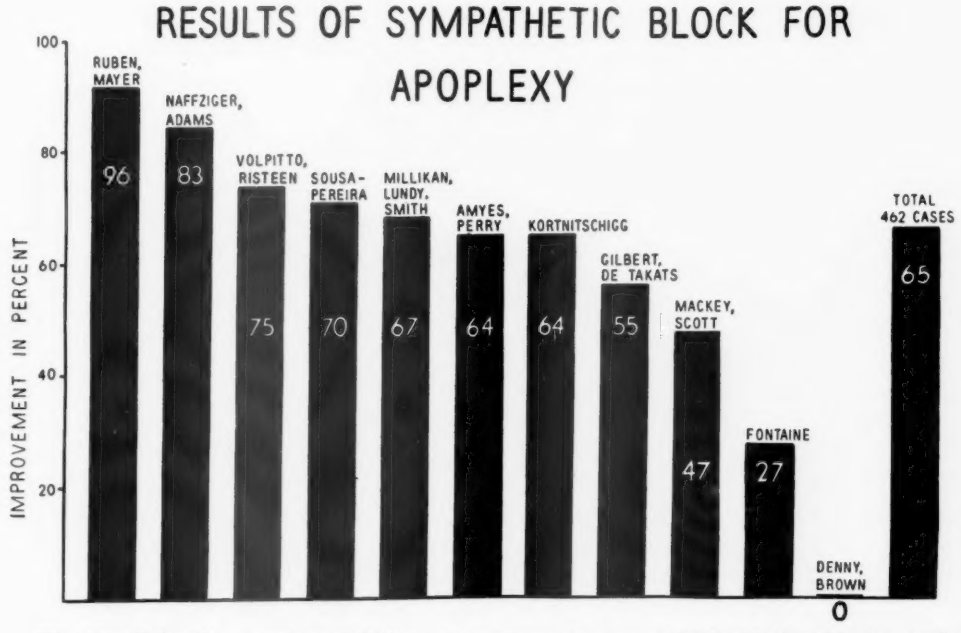


Fig. 8.—Note the great variability of response to sympathetic block; 96 per cent improvement in the cases of Ruben and Mayer, to no improvement in Denny-Brown's observations. The first 2 cases of Leriche, both successful, are not included. Of a total of 462 patients, 302 (65 per cent) were reported as obtaining benefit. Our own series shows a somewhat lower percentage of improvement.

These may well be the reasons why results are only obtained in about one-half of the injected cases. In a recent survey of the available literature, we found that of a total of 462 cases 302 (65 per cent) obtained benefit, whereas 160 (35 per cent) were failures (Fig. 8). "Good" results were listed with the excellent ones under improvement, whereas "dubious" ones were listed with the failures. Since this chart was prepared,[2] an additional group of 71 patients (Bohmert, Principe, and Gatenby)[21-23] showed a 66 per cent improvement, thus not appreciably influencing the over-all percentage of success. The percentage of success calculated from the reported cases in the literature is higher than

our own, owing to two highly favorable reports on the left hand of the chart. It is quite possible that an author, having tried six or ten successive cases without benefit, will abstain from further trial and not report his negative cases. Our first case was so dramatic that we did not become discouraged when a negative series of six patients followed the first positive result.

COMMENT

How should one evaluate then, at present, this simple, harmless procedure in the treatment of strokes? The exact localization, the amount of ischemic necrosis, in fact, even the nature of the vascular accident being as uncertain as they are, I have regarded this method more as a diagnostic procedure. For if a rapid regression of neurological symptoms follows promptly after a cervical sympathetic block, one can be sure that improvement will continue unless hemorrhage, tumor, or later thrombotic episodes vitiate the initial result. With the advent of more active surgical measures, including angiograms, carotid embolectomies, evacuation of intracerebral clots, all of which require mature neurosurgical judgment, further improvement may be expected in the future. At present, the overwhelming majority of apoplexies are due to cerebral infarcts, ischemic or hemorrhagic, whether they are primarily due to emboli, to thrombi, or to cerebrovascular insufficiency in cerebral arteriosclerosis. As long as all of these lesions create a perifocal edema of smaller or larger extent, sympathetic blocks seem worthy of further trial. For the rare causalgic state after apoplexy, cervical sympathetic block may be worth trying months or years after the accident.

SUMMARY

Fifty-five patients have received from one to ten cervical sympathetic blocks as part of an emergency treatment of apoplexy. A little over half of these patients and 65 per cent of 533 patients collected from the literature have shown immediate improvement after this simple procedure. There seems to be, at present, no way of selecting the suitable case, but the technique advocated is harmless and the results, when obtained, are gratifying. The prognostic value of the block may be the most significant aspect of the procedure.

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CLINICAL GRADING OF THE SEVERITY OF OBLITERATIVE ARTERIAL DISEASE OF THE LOWER EXTREMITIES

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THE lower extremity can remain viable despite a drastic reduction in the volume of its blood flow. Precise information as to the exact amount necessary to maintain viability is not available; but a personal estimate, believed to be conservative, is that it is less than 10 per cent of the normal resting blood flow. Thus, a wide span exists between the limb in which some evidence of arterial insufficiency can be detected by the examining physician and the limb with impending or actual gangrene. An attempt has been made to divide this span into stages indicating increase in grade of arterial insufficiency. For this clinical evaluation, only those symptoms and signs considered to be least susceptible to misinterpretation are utilized. It is recognized that clinical methods of quantitating the degree of arterial insufficiency present in a limb are indirect and crude, but it would seem that any reasonably reliable method of classifying patients with this common condition would serve to facilitate the selection of the most appropriate form of treatment for a given patient and would help the physician to evaluate results of therapy more precisely.

The method of grading presented applies to extremities with chronic obliterative arterial disease due to arteriosclerosis or thromboangiitis obliterans. It cannot be used to evaluate an extremity immediately following an acute occlusion of a major artery.

A summary of the suggested grading is shown in Table I.

Grade I. A Decrease in Amplitude of the Dorsalis Pedis and Posterior Tibial Pulse.—The diagnosis of obliterative arterial disease cannot be made by this finding alone unless there is a marked difference between the amplitude of the pulse in the two extremities. In most instances, the diagnosis will have been made by a history of intermittent claudication. Although physicians usually grade the amplitude of a peripheral pulse from zero to 4 plus, it is suggested that for ease of grading the pulse be considered "normal," "diminished," or "absent."

Grade II. Absence of the Dorsalis Pedis and Posterior Tibial Pulse.—Silverman[4] reported on the incidence of palpable pulsations in the feet of normal adults. His examination of 908 white soldiers revealed that the dorsalis pedis pulse was absent in 14.5 per cent of the left lower extremities and in 12.6 per cent of the right lower extremities. The posterior tibial pulse was absent in 2 per cent of the left and in 2.2 per cent of the right lower extremities. The

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

absence of both the dorsalis pedis and posterior tibial pulse was noted in only 0.3 per cent of the left and in 0.2 per cent of the right lower extremities. In 106 Negro soldiers, the dorsalis pedis pulse was absent in 4.7 per cent of the left, and in 1.9 per cent of the right lower extremities; the posterior tibial pulse was absent in 8.5 per cent of the left, and in 7.5 per cent of the right lower extremities. Absence of both the dorsalis pedis and posterior tibial pulse was not observed in the Negro soldiers examined. Thus, the absence of both the dorsalis pedis and posterior tibial pulse in a given extremity is sufficient evidence for a diagnosis of arterial insufficiency in almost all instances. The degree is considered Grade II.

TABLE I. GRADES OF ARTERIAL INSUFFICIENCY

I.	Decrease in amplitude of pulse in foot
II.	Absence of posterior tibial and dorsalis pedis pulse
III.	Absence of popliteal pulse; absence of posterior tibial and dorsalis pedis pulse in a patient with diabetes mellitus
IV.	Rest pain in the foot; rubor on pendency Pallor of toes on elevation Absence of moisture
V.	Continuous pain in foot; petechial hemorrhages of the dorsum of the foot; skin necrosis

For example, F. K., a 49-year-old white man examined in August, 1953, complained of pain in both calves on walking six or eight blocks which was relieved by standing still. There was no rest pain. The dorsalis pedis and posterior tibial pulses were absent bilaterally. The popliteal pulse was present bilaterally. He was advised to stop smoking; he had been averaging 30 cigarettes a day. He believes that he can walk a greater distance now, and at a faster rate, than two years ago. He has no other complaints and works daily.

Grade III. Absence of the Popliteal Pulse and *Absence of the Dorsalis Pedis and Posterior Tibial Pulse in a Diabetic Patient.*—The popliteal is believed to be the most significant single pulse for evaluating a limb with arterial insufficiency. Wessler and Silberg[5] noted that it was present in only 13 per cent of a series of limbs with arterial insufficiency that came to amputation and was present in 53 per cent of a control series in which amputation was not necessary. The femoral pulse, on the other hand, was present in 94 per cent of both the amputated extremities and those in the control group. Unfortunately, the popliteal pulse is the most difficult to feel, especially if one tries to palpate it with the patient in the sitting position. However, its presence or absence can be determined in most instances if the patient is examined when lying supine and the knee is flexed passively. In doubtful cases, an oscillometer or an aneroid sphygmomanometer may be used to check for its presence.

Clinically, some differences are noted between extremities with obliterative arterial disease in patients having diabetes mellitus and those without this disease. One point of difference is that the diabetic patient is likely to have greater involvement of the distal arteries and arterioles than the nondiabetic patient. For example, it is not uncommon to encounter a diabetic patient with a bounding popliteal pulse and gangrene of the foot, and occasionally one is observed with a strong posterior tibial and dorsalis pedis pulse who has at the same time ischemic necrosis of the toes. On the other hand, a nondiabetic patient with ischemic necrosis of the foot will usually have obliteration not only of the dorsalis

pedis and posterior tibial pulse but also of the popliteal pulse. This difference appears to be a real one and is of sufficient magnitude to include in Grade III diabetic patients with absent posterior tibial and dorsalis pedis pulses. The tendency for greater involvement of the arterioles and small arteries in the diabetic patient with arteriosclerosis may explain, in part at least, why it is possible more often to perform a successful amputation of one toe or a transmetatarsal amputation in a diabetic patient than in a patient with arteriosclerosis without diabetes.

As an example of a patient with Grade III arterial insufficiency without diabetes, there is G. McF., a 55-year-old white man who was seen on May 10, 1954, complaining of pain in both calves on walking, of three or four months' duration. Pain was relieved by standing still and recurred when he walked again. There were no other symptoms. Elimination of smoking (he had been averaging 40 cigarettes a day) and the use of a vasodilating agent orally did not relieve his symptoms. In January, 1955, a left lumbar sympathectomy was performed. Postoperatively, he has noticed a significant improvement in the general well-being of the limb. The temperature of the left great toe is 89.6° F. after equilibration in a room at 68° F. for one hour; and at the same time the amplitude of the pulse in the left second toe is two and one-half times what it was under the same conditions preoperatively. He believes he can walk a little farther than he could before the operation.

To this point, the presence or absence of palpable pulse has been the criterion for estimating the degree of arterial insufficiency in the extremity. Although a palpable pulse is a highly useful sign of arterial adequacy in the lower grades of arterial insufficiency, it is not an absolute criterion for the higher grades for the following reasons: (1) it tells one nothing of the caliber of the lumen just distal to the point at which it is palpable; and (2) its absence does not necessarily mean that the artery is nonfunctioning. For, just as a vein, an artery may serve as a conduit for a large volume of blood while not pulsating perceptibly. To illustrate, the femoral pulse will be nonpalpable if the aorta or common iliac artery is completely occluded; but an arteriogram or surgical exploration may demonstrate the fact that it is functioning.

For these reasons, the nutritional state of the integument of the foot and toes as judged by physical examination appears to be a better criterion for estimating the degree of arterial sufficiency in an extremity with a high grade of obliterative arterial disease.

Grade IV. The Presence of Rest Pain in the Foot and/or Rubor of the Foot on Pendency; Pallor of the Foot on Elevation; Extreme Dryness of the Foot.—Rest pain is quite specific although frequently confused with muscular cramps occurring in the presence of venous insufficiency or muscular hyperirritability. Rest pain in the foot occurs in the great toe or the region of the head of the first metatarsal bone most frequently but may occur elsewhere on the distal portion of the foot. At first, it is likely to occur after the patient has been asleep for several hours, but in the more advanced stages of arterial insufficiency it begins as soon as the limb is elevated on the bed. When the patient sits up and permits the limb to be pendent, the pain is partially or completely relieved. This pain is frequently ascribed to ischemic neuritis. The close relationship of the position of the limb to the presence of pain suggests that it may be due, in part at least, to emptying of the capillaries of the distal portion of

the foot. When the foot is elevated, the small trickle of blood reaching the capillaries through arteries and arterioles whose lumina are greatly reduced in size, is rapidly drained off through veins whose lumina are still patent. When, however, the limb is pendent, the gravitational force helps to keep the capillaries filled with blood.

Rubor may vary in depth from a faint pink-red to a vivid red or red-purple. The latter is familiar to all physicians. When the color is very faint, it may be difficult to distinguish rubor from the normally flushed skin. In most instances, however, it can be detected by comparing the color of the two feet, and observing the changes in color that take place when the feet are elevated and then permitted to be pendent.

Pallor of the toes on elevation of the limb is a highly significant sign of arterial insufficiency but the varying degree of pallor that may be observed is difficult to interpret. Lewis[3] has pointed out that the pink hue of skin is due to the presence of blood in the subcapillary venous plexus and not in the arterioles or capillaries as is frequently supposed. This is why some pallor may be noted when a normal limb is elevated above heart level and venous drainage is augmented. The degree of pallor is accentuated when arterial insufficiency is present but to correlate the two becomes a matter of personal judgment. One method of solving the difficulty and making pallor a useful criterion from the practical point of view is to take it into consideration only when present in the extreme degree, that is, if all the pink color fades when the limb is elevated for thirty seconds and only a lemon-yellow or cadaveric color remains. When this degree of pallor exists, the limb is usually in the fourth stage of arterial insufficiency.

Similarly, extreme dryness of the foot is a very helpful sign although not always easy to interpret. A normal extremity may be perfectly dry when the environment is cool. But if the environment is warm and the skin of the opposite foot or of the legs is moist, then extreme dryness of the foot is a highly significant sign of arterial insufficiency and suggests, furthermore, that the arterioles of the foot cannot be made to dilate either by medicinal agents or by sympathetic ganglionectomy.

The presence of rest pain or rubor is considered sufficient evidence for placing a limb in Grade IV regardless of the presence or absence of peripheral pulsations. Pallor of the foot on elevation and extreme dryness are highly suggestive but not absolute signs of this degree of arterial insufficiency.

An example of Grade IV arterial insufficiency is J. H. P., a 75-year-old white man seen in November, 1953, with intermittent claudication, rest pain, coldness, numbness, and tingling of the left lower extremity. A weak pulse was palpable in the superficial femoral artery but none below this point. There was 4 plus pallor on elevation and rubor of the toes and dorsum of the foot on pendency. The skin of the foot was completely dry. During the next eighteen months, the rest pain disappeared and the other symptoms, except intermittent claudication, improved. The pulsations are unchanged, pallor is not complete, and rubor is absent. Treatment consisted of instructions on the proper care of the feet and Buerger-Allen exercises. The possibility that the improvement was entirely spontaneous is a real one.

Grade V. Continuous Pain in the Foot; Petechial Hemorrhages in the Skin of the Dorsum of the Foot; Skin Necrosis.—When rest pain becomes so severe

that the patient no longer obtains relief by permitting the limb to be pendent and when the pain becomes constant, the limb is considered to be in the fifth grade of arterial insufficiency. It is at this point that swelling of the foot and ankle, which was not previously present, may be seen. This gravitational edema appears to be due to the fact that the limb is maintained in the pendent position constantly. It provides additional evidence that the ischemia is reaching an irreversible stage.

In some limbs with marked rubor on pendency, small petechial hemorrhages are seen on the dorsum of the foot. This, too, is evidence of the most advanced form of obliterative arterial disease.

If a patient has gangrene of a toe or part of the foot, he is, obviously, in this group. Some patients are encountered who have discrete areas of skin necrosis of the foot or leg. This is believed to be due to obliteration of the smallest arteries and arterioles, as suggested by Edwards.[2] Although some limbs remain viable despite the presence of localized areas of skin necrosis, and some such areas heal spontaneously, nevertheless their presence usually indicates that the limb is in the most advanced stage of obliterative arterial disease.

C. F., an 86-year-old white woman with diabetes mellitus, was admitted to Good Samaritan Hospital in January, 1954, complaining of severe pain in the toes and dorsum of the right foot. She had necrosis of the tip of the second and third toe, rubor on pendency, 4 plus pallor on elevation, and petechial hemorrhages of the dorsum of the foot. Pain in the toes and in the foot was constant, although slightly relieved when she permitted the foot to hang over the side of the bed. The femoral and popliteal pulses were present and strong bilaterally. No pulse could be felt in either foot. A below-the-knee amputation was performed but failed to heal, and a supracondylar amputation had to be carried out.

A suggested application of this method of grading to therapy is as follows:

Grade I: The patient is given general instructions on foot hygiene and advised to avoid extremes of cold or heat and to eliminate the use of all forms of tobacco.

Grade II: Subjective and objective improvement in the limb is expected following lumbar sympathectomy. Even intermittent claudication may be improved in extremities in this group. If surgical treatment is contraindicated, long-term treatment with a peripheral vasodilating agent may be considered.

Grade III: Most extremities without arterial insufficiency due to a localized obstruction in the terminal aorta, iliac, or femoral artery will fall into Grade III of this classification. In this instance, restoration of continuity of the major artery should be considered. Otherwise, lumbar sympathectomy is believed to be the treatment of choice. Even though the ability to walk may be unimproved, the patient will notice the increased warmth in the foot and will almost always say that the limb feels much better than it did prior to operation. Indeed, all clinical evidence indicates that these extremities are benefited significantly by lumbar sympathetic ganglionectomy. Final proof, however, that lumbar sympathectomy will delay or prevent gangrene in the extremity is not available at this time. One reason for this is that, contrary to popular opinion, patients with obliterative arterial disease secondary to arteriosclerosis may get along very well for years without specific treatment of any type. Spontaneous im-

provement may occur in any stage of obliterative arterial disease although, of course, it is more likely to occur in extremities with lesser degrees of arterial insufficiency. This spontaneous improvement also accounts for the difficulty in evaluating the result of long-term medical therapy in these patients. The sudden striking changes that occur in a limb with Grade III arterial insufficiency following lumbar sympathectomy are not observed following administration of vasodilating agents orally. And, if there is gradual improvement of the extremity over a long period of time while the patient is receiving a vasodilating agent, it becomes almost impossible to determine whether or not the improvement is spontaneous or is due to the medication being used.

Grade IV: Subjective and objective improvement of extremities in this group following lumbar sympathectomy are much less striking than in extremities in Grade III and, as in the case of Grade III extremities treated with vasodilating agents, even if the limb improves following operation it is difficult to be certain that the beneficial effect is due primarily to the ganglionectomy. Final proof of the value of lumbar sympathectomy of any type of medication in the prevention of irreversible changes in these extremities must await long-term observations on the incidence of gangrene in extremities in this group with and without treatment.

COMMENT

An explanation as to why intermittent claudication is not included in this classification is indicated. Intermittent claudication may be defined as pain (or tiredness) in a muscle group that occurs only after repeated contraction of the muscle and is relieved promptly when the muscle is rested. Typically, it is experienced after a patient walks a short distance and is relieved by standing still. Each time the patient walks a similar distance, the pain recurs. It is not necessary to elevate the extremities for relief as may be the case in extremities with venous insufficiency. This is one of the most specific symptom-complexes in medicine and indicates, unequivocally, that the muscle is not receiving sufficient nourishment to carry out its normal function. For practical purposes (with the exception of extremities with an arterial injury or the rare instance of severe anemia), it always indicates obliterative arterial disease. It establishes the diagnosis and all that remains to be determined is the degree of arterial insufficiency present. From a qualitative point of view then, it is the most important symptom in peripheral arterial diseases. Quantitatively, however, it can be a source of confusion. There are three major reasons for this.

The walking rate of a patient with obliterative arterial disease has a profound effect upon the total distance he is able to walk. For example, it was observed that the total walking distance might be doubled by reduction of the rate of walking from 120 to 100 steps per minute.[1] In laboratory studies, one is able to control the rate of walking, to force the patient to maintain a constant cadence, to provide level ground, and to measure the distance accurately. But one cannot obtain this type of information from the patient. Therefore, to rely on the patient's estimate of his ability to walk before or after any type of treatment may introduce serious error.

A second point is that the correlation between the severity of muscle ischemia, as judged by the distance a patient can walk without pain, and integumental ischemia is not always good; and, as a matter of fact, may be very poor. It is not unusual to encounter a patient with a localized obstruction in the terminal aorta or iliac artery who has severe intermittent claudication, and yet who has no evidence of malnutrition of the skin. On the other hand, as mentioned previously, a patient may be seen with gangrene of the toes or foot who has a bounding popliteal or even a posterior tibial and a dorsalis pedis pulse who has never had intermittent claudication. Therefore, arterial insufficiency of the muscles of an extremity must be considered separately from arterial insufficiency of the integument; and, since a reliable clinical method of grading muscular insufficiency is not available currently, it seems best to exclude intermittent claudication as a quantitative measure of arterial insufficiency of the extremity as a whole.

Finally, intermittent claudication is never in itself an indication for amputation even though it can be very disabling. Therefore, a decrease in or absence of the peripheral pulse and the effect of diminished blood supply on the skin appear to be better criteria for making a quantitative estimate of the degree of arterial insufficiency present in an extremity.

Fundamentally, the most important determinant of the result of therapy for extremities with obliterative arterial disease is the degree of arterial insufficiency present at the onset of treatment. And, since the severity of arterial insufficiency in the extremity may vary from a degree which causes only mild discomfort when the patient willingly accepts amputation for the relief of pain, and since the span between these extremes is very wide, it is definitely advantageous to have a system of grading the severity of the disease found on clinical examination that will aid in selecting the most appropriate form of therapy and evaluating therapeutic results. In formulating this method, the aim has been to combine, as nearly as possible, simplicity with reasonable comprehensiveness. Other symptoms and signs have been excluded, not because they are considered unimportant, but because their inclusion would defeat the purpose of simplicity and discourage widespread adoption of the method.

Other determinants such as specific therapy and ancillary methods including rest of the part, control of infection, avoidance of heat, and so forth, influence therapeutic results profoundly; indeed, to such a degree that each merits separate consideration. However, at this time, it is believed best to stress the most fundamental determinant, that is, the severity of the disease at the onset of therapy.

SUMMARY

The functional reserve capacity of the arterial circulation of the lower extremity is so great that it is estimated that less than 10 per cent of the normal resting blood flow can sustain life in the limb. Thus, in extremities with chronic obliterative arterial disease, a wide span exists between the limb in which some evidence of arterial insufficiency can be detected by the examining physician and

the limb with impending or actual gangrene. A clinical method of dividing this span into five stages of increasing severity of the disease is presented, together with suggestions for therapy in each of the five stages.

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THE LABORATORY ASSESSMENT OF THE DIGITAL CIRCULATION

A Comparison of Methods

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INTRODUCTION

RECENT advances in vascular surgery have emphasized the need for methods of assessing the efficiency of the peripheral circulation. Much can be learned by clinical examination and indeed this is frequently sufficient in the average case. Complete evaluation of the status of the peripheral circulation and of its reserve, however, requires precise measurement of the blood flow in the part in question and of the functional changes of the circulation in response to different experimental conditions. In the past, studies of changes in the temperature of the skin, plethysmography, and calorimetry were most commonly used for this purpose. More recently, radioactive isotope techniques have been applied to investigation of the peripheral circulation as they have in other fields of medicine.[1]

On review of the literature, several studies of the peripheral circulation were found, correlating variations in the temperature of the skin with blood flow[2] and with isotope build-up or clearance.[3] These three modalities, however, have not been simultaneously compared in the same individual. Furthermore, the digital circulation, a convenient vascular bed representative of acral skin, has not been studied by isotope techniques. It was thought appropriate, therefore, to test these three methods as applied simultansously to the digital circulation in the same individual. The experiments to be reported were designed to permit a comparison of the three methods under the following conditions: (A) arterial occlusion and release; (B) venous congestion and release; (C) reflex vasoconstriction; and (D) reflex vasodilation. These were chosen because they were thought likely to give the most information with regard to sensitivity to change, magnitude of response, and reproducibility. It was hoped that the data obtained would permit assessment of the relative merits of the three methods.

METHODS

Apparatus.—A "well" type scintillation counter (Fig. 1), heavily screened circumferentially and protected on its upper surface by a 1.5 cm. lead plate, was supported on its side on a table beside the examination bed. The counter was connected with a counting rate meter, and this in turn with an Esterline-Angus recording milliammeter. The volume of the "well" within the crystal of the counter was such that an approximately linear response could be obtained with volumes of radioactive material up to 5 ml. The "well" was adapted as an oncometer, as illustrated in Fig. 1, and blood flow was measured by venous occlusive

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

plethysmography. Approximately 8.5 ml. of finger, comprising the middle and distal phalanges, could be introduced into the "well" oncometer. A small-bore pressure tube led from the oncometer to a strain gauge, and recordings were made as previously described.[4] Skin temperature was recorded by a Brown automatic potentiometer using an iron constantan thermocouple. The apparatus was set so that skin temperature was marked on a moving chart every thirty seconds. Room temperature was recorded by Minneapolis-Honeywell electrical thermometers.

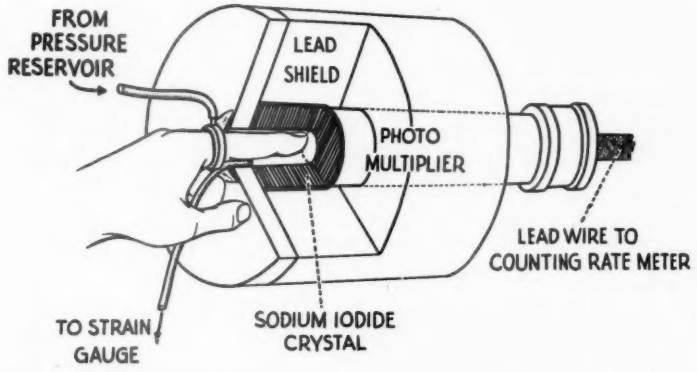

Fig. 1.—A diagrammatic representation of the scintillation counter adapted for use as an oncometer.

Preparation.—Eleven subjects of both sexes, with or without occlusive vascular diseases, were studied in a constant temperature room maintained at 20° C. or deliberately varied during the course of the observations. The data differed only quantitatively, and not qualitatively, between the subjects with and without peripheral vascular disease. I^{131}-labeled albumin had been chosen as the most suitable tracer material for this study. In consequence, six hours before the projected time of the experiment, the subjects were all given 15 minims of Lugol's iodine solution to prevent appreciable uptake of I^{131} by the thyroid gland. The patient rested comfortably on a bed with only the pelvis and chest covered by thin towels. The arm to be studied was supported on a pillow so that the finger could be easily inserted into the oncometer. The scintillation counter was arranged parallel to the body, allowing maximal advantage to be taken of its lateral shielding, and with its axis approximately 15 cm. above the sternal level. The index finger was usually selected for investigation and was fitted with an appropriately sized ring occlusive cuff. This finger was then inserted into the oncometer beside the recording pressure tubing and both were sealed in with sealing compound. A thermocouple was lightly applied to the pulp of the ipsilateral third finger and a sphygmomanometer cuff placed around the arm just above the antecubital fossa. The hand and arm were arranged so that they lay in a position of maximum shielding from the axis of the well. This reduced the amount of radiation entering the counter through the opening in the lead cover plate occupied by the finger. The finger was steadied in the oncometer by applying broad longitudinal (but not circumferential) strips of adhesive tape along the forearm and on to the outside of the counter.

When skin temperature had equilibrated with that of the room and the oncometer proved airtight, a vein of the free arm was injected with 50 to 100 μc of I^{131}-labeled serum albumin* in 1 to 2 ml. of saline solution. Thereafter, blood flow tracings were taken approximately every three minutes, or more often in some parts of the experiment. Circulatory radioactivity and skin temperature of the third finger pulp were recorded automatically.

Procedure.—Studies of the effects of arterial and venous occlusion and release upon the reflexly vasoconstricted hand were started about fifteen minutes after injection of the radioactive solution, when the Esterline-Angus tracing was steady. At this time, the sphygmomanometer cuff was inflated to occlude arterial inflow. Five minutes later, the pressure

*Obtained from Abbott Laboratories.

within the cuff was released and blood flow records were taken until the reactive hyperemia had subsided. The same procedure was followed after the application of a venous occluding pressure of 70 mm. Hg for five minutes. The hand was subsequently reflexly vasodilated by immersing the legs in water at 45° C. until skin temperature had stabilized. Observations were made on the sensitivity of the three methods of investigation in reflecting the changing digital circulation as vasodilation progressed. Studies of arterial and venous occlusion were then repeated as described for the vasoconstricted hand. The room temperature was maintained at 20° C. throughout all the experiments, except in three cases when it was raised during the period of reflex vasodilation to a maximum of 29.5° C. This enhanced the reflex vasodilating effect but reduced the usefulness of the skin temperature data. In some cases the whole study was begun with the patient warm and the skin vasodilated. The patient's hand was then reflexly vasoconstricted by immersing the legs in water at 10° C. The sensitivity of the three methods for showing changes in the circulation as vasoconstriction progressed was studied as it was for vasodilation. The finger was then removed from the oncometer, and measurements of background radioactivity were made with the hand resting in the position it had occupied, but without the finger in the oncometer. The volumes of the finger under examination and of the oncometer were then measured, so that plethysmographic data could be converted to standard values.

All measurements of blood flow were recorded in terms of milliliters of blood per 5 ml. of digit per minute. Radioactivity was recorded as milliamperes, taken from the Esterline-Angus milliammeter, 1 ma. being equivalent to 5,000 counts per minute. The temperature of the skin was recorded in degrees centrigrade.

RESULTS

In the presentation of the results obtained in this study, plotted blood flow estimations are averages of up to 10 plethysmographic "slopes." The digital radioactivity in response to the experimental conditions was calculated by subtracting from the total radioactivity that recorded as "background" at the end of the experiment. This background, which was calculated only at the end of each experiment, consisted of the usual level added to that referable to the proximity of the hand and body to the counter. Since some of the values so obtained were actually negative, one must conclude that the background varied considerably in the course of the experiment. The working amount of variation in radioactivity during any experiment was, on the average, 50 per cent of the total recordable radioactivity during reflex hyperemia in the vasodilated hand.

Responses to Arterial and Venous Occlusion and Release.—

A. Vasoconstricted hand: Figs. 2 and 3 show the relationship between the three entities studied when the hand was vasoconstricted before, during, and after the stimulus of arterial and venous occlusion applied for five minutes, respectively. The fall of radioactivity during the period of arterial occlusion is probably due to drainage of blood from the finger into the veins of the dependent arm. The rise of radioactivity during the corresponding period of venous occlusion is due to congestion in the finger and hand. After arterial occlusion, all three values are above control levels, representing a state of reactive hyperemia. Release of the venous congesting cuff, however, returns all values to nearly control levels, within two minutes, and there is little evidence of a reactive hyperemia. This may be due to the distance of the congesting cuff from the finger having a delayed and therefore lessened effect on the digital

vascular bed, and to the low environmental temperature. The lack of increased radioactivity, and indeed the slight fall below the control levels, may be offered as good evidence that the radioactive tracer material has remained for the most part in the blood stream and has not equilibrated in the extravascular fluid during the venous congestion. The decrease of skin temperature during the period of venous occlusion (Fig. 3) indicates a decreased rate of blood flow.[5, 6] The former has not returned to the control level within two minutes of release of the occlusion.

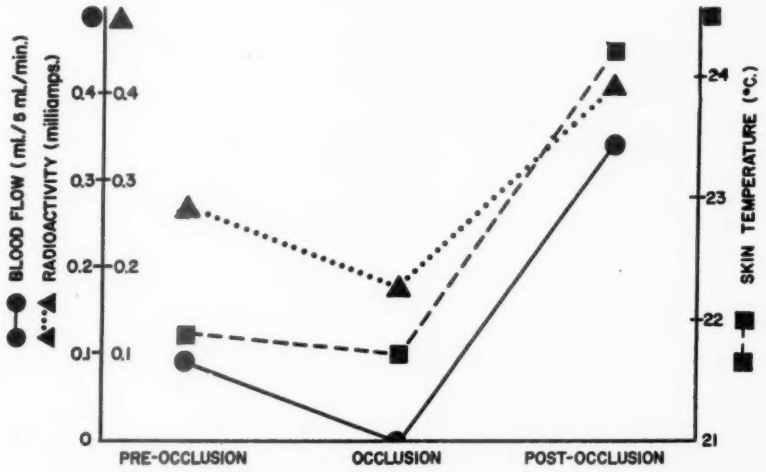

Fig. 2.—The points plotted are average values recorded during a two-minute period immediately preceding arterial arrest for five minutes (*preocclusion*); during a similar period preceding the release of arterial arrest (*occlusion*); and directly after release of the arterial tourniquet (*postocclusion*). The high postocclusion values are indicative of reactive hyperemia. In this and all subsequent graphs, the unit of radioactivity of 1 ma. is equivalent to 5,000 "counts" per minute.

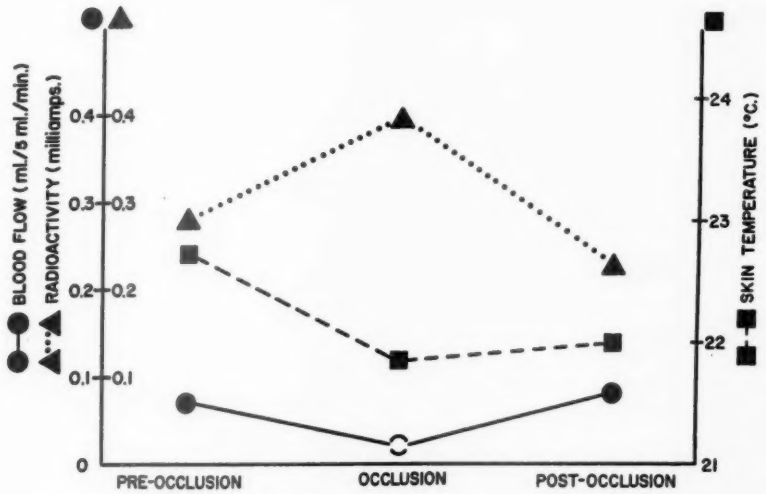

Fig. 3.—The points plotted are average values recorded during a two-minute period immediately preceding venous occlusion for five minutes (*preocclusion*); during a similar period prior to release of venous occlusion (*occlusion*); and immediately after release of venous occlusion (*postocclusion*). The cuff pressure used for venous occlusion in the arm was 70 mm. Hg. No significant reactive hyperemia was produced by this degree of venous occlusion. There is no evidence of diffusion of radioactive material into extravascular tissues during the period of venous occlusion.

B. The vasodilated hand: Figs. 4 and 5 give the corresponding values when the finger was reflexly vasodilated before the experiment. Now the reactive hyperemia is not demonstrated by a rise of skin temperature as it was when the experiment was performed for the vasoconstricted hand; nor is the

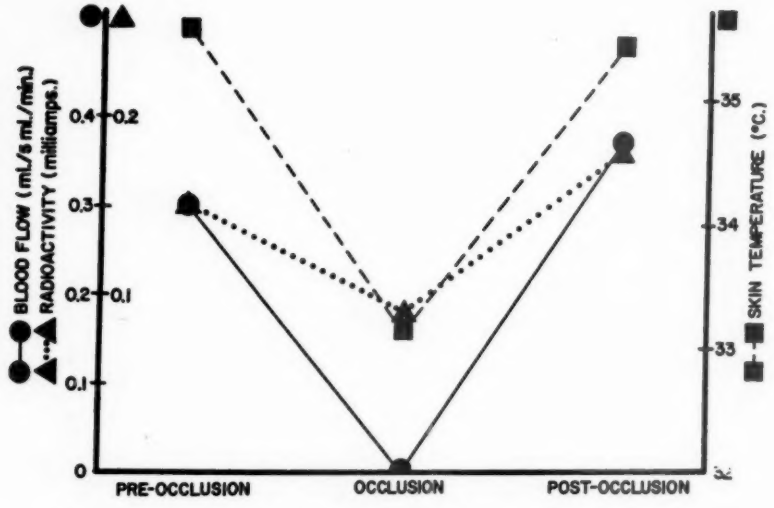

Fig. 4.—The points plotted are average values obtained as in Fig. 2, except that the hand was in a state of reflex vasodilation instead of reflex vasoconstriction. There is no increase of skin temperature above the vasodilation level during the postocclusion period. There is an increase in the rate of blood flow above the preocclusion vasodilation level. There is a similar increase in the level of radioactivity, but this may represent an increased extravascular radioactivity in this particular experiment (see text).

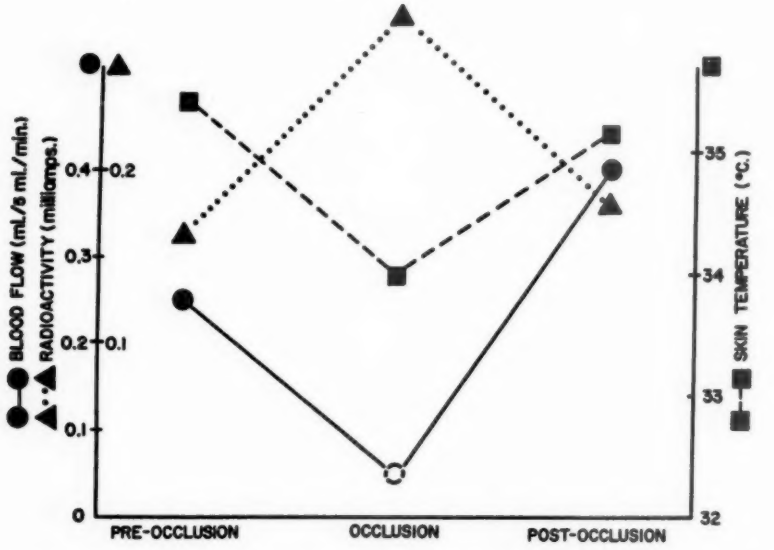

Fig. 5.—The points plotted are average values obtained as in Fig. 3, except that the hand was in a state of reflex vasodilation instead of reflex vasoconstriction. During occlusion, increased radioactivity and decreased skin temperature are similar to but more marked than those with the hand initially vasoconstricted. There is an obvious increase in the rate of blood flow upon release of venous occlusion, but this is not reflected in the temperature of the skin or in the level of radioactivity.

radioactivity significantly increased beyond the level reached by simple reflex vasodilation. The rate of blood flow, on the other hand, is increased well above the maximal level attained by reflex vasodilation alone.

The histogram of Fig. 6 relates digital blood flow, circulating radioactivity and skin temperature in one experiment during the initial period of vasoconstriction, during reflex vasodilation, and during reactive hyperemia superimposed upon reflex vasodilation. It can be seen from the last two groups of columns that although blood flow through the finger increased still more during the superimposed reactive hyperemic phase, circulating radioactivity did not do so, nor was skin temperature further elevated.

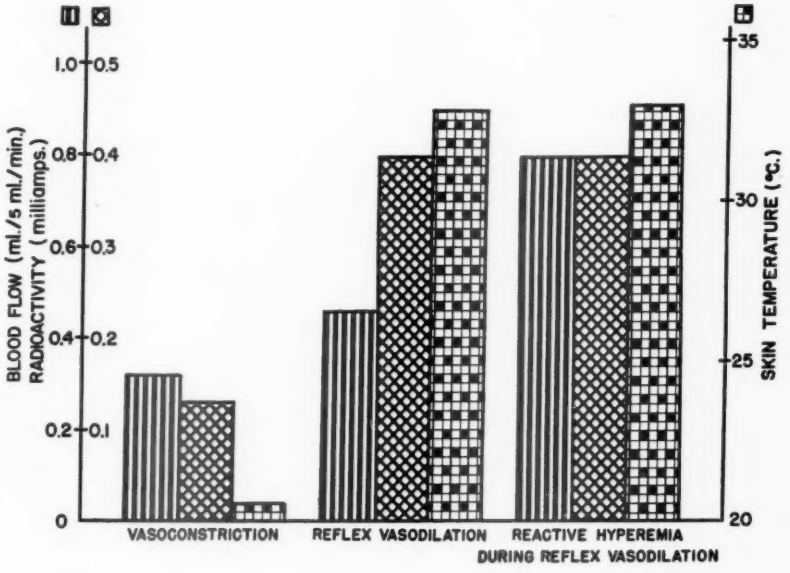

Fig. 6.—The histogram illustrates equilibration levels of digital blood flow, radioactivity, and skin temperature under three experimental conditions. The rate of blood flow increases beyond the level reached with simple reflex vasodilation when reactive hyperemia is superimposed; skin temperature and radioactivity do not.

The inability of increase in blood flow to raise skin temperature above a point determined by the resultant of blood and ambient temperatures is shown for finger and toe in Figs. 7 and 8, respectively. The data were obtained from normal subjects and from patients with peripheral vascular disease. Each point represents the maximal value attained in one subject. Both graphs relate to ambient temperatures of 20° C. It can be seen that when digital blood flow has reached a rate of approximately 1 ml. per 5 ml. of part per minute, further rise of skin temperature is negligible.

Rate of Response to Arterial and Venous Occlusion and Release.—Figs. 9 and 10 show the courses of the responses to arterial occlusion and subsequent release in the vasoconstricted and reflexly vasodilated hand, respectively. There is a qualitative relationship between the three variants in their responses to the tests imposed. During the period of arterial occlusion, radioactivity falls in both the vasoconstricted and vasodilated hands. This fall is not a reflection of

cessation of blood flow but rather the result of drainage of blood from the finger into the dependent veins of the arm by gravity. Skin temperature does not fall in the vasoconstricted hand because the hand was already nearly in equilibrium with room temperature, but does fall in the vasodilated hand.

Upon release of the arterial occlusion there is an immediate increase in the rate of blood flow far greater than the control rate of both the vasoconstricted and vasodilated hands. After release of the arterial tourniquet radio-

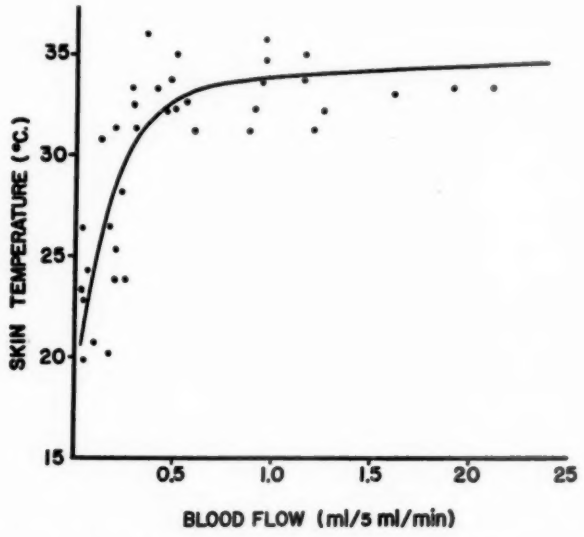

Fig. 7.—This graph relates the temperature of the digital skin to the rate of digital blood flow for the finger. The points are maximal values attained in each case during reflex vasodilation. Increase of digital blood flow over 1 ml. per 5 ml. of part per minute has very little effect upon the skin temperature.

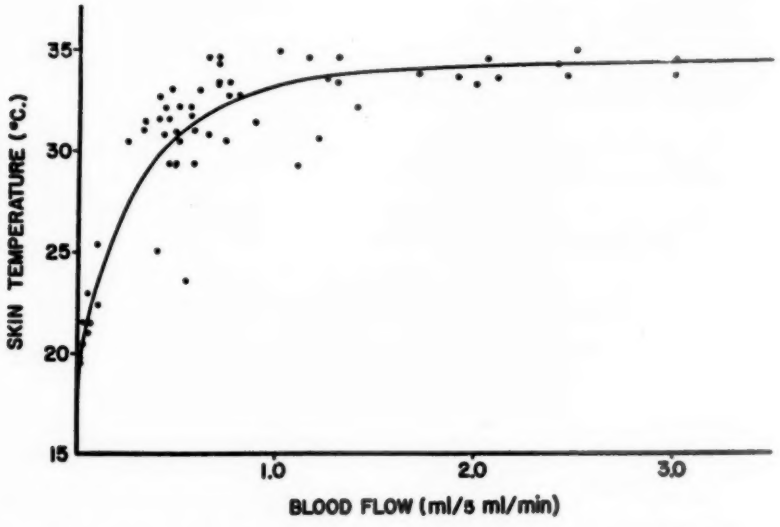

Fig. 8.—This graph relates the temperature of the digital skin to the rate of digital blood flow for the toe. The data were obtained and plotted as in Fig. 7. In correspondence with the finger, increase in the rate of blood flow over 1 ml. per 5 ml. of part per minute has very little effect upon the temperature of the skin.

activity also increases about as rapidly as the increase in the rate of blood flow and in the vasoconstricted hand rises well above the control preocclusion level. In the vasodilated hand, however, the radioactivity does not appreciably surpass the control level. In the same hand the skin temperature also rises rapidly to its maximum after release of the arterial tourniquet, but not significantly

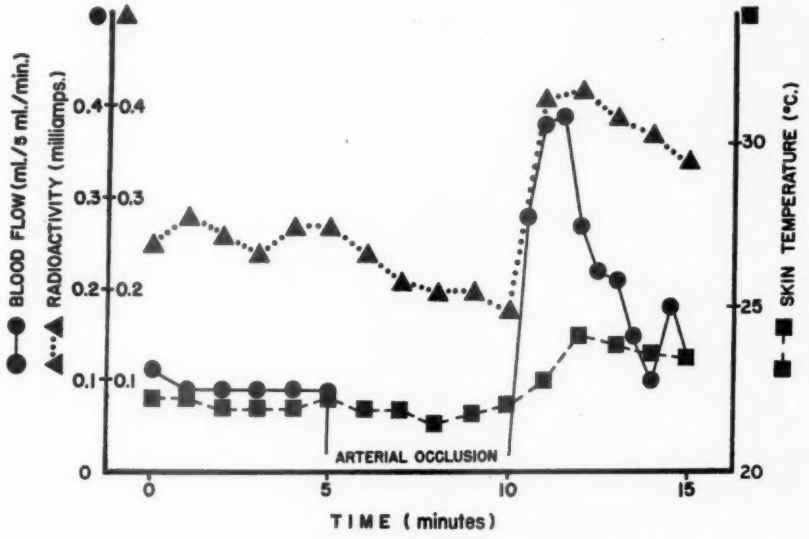

Fig. 9.—The points on the three graphs represent averages of determinations made during one-minute periods before, during, and immediately after release of arterial occlusion. Reactive hyperemia is demonstrated by all three techniques, but least readily by skin temperature changes. Note the slow return of radioactivity from its maximal level, possibly attributable to diffusion of radioactive protein into the tissues (see text).

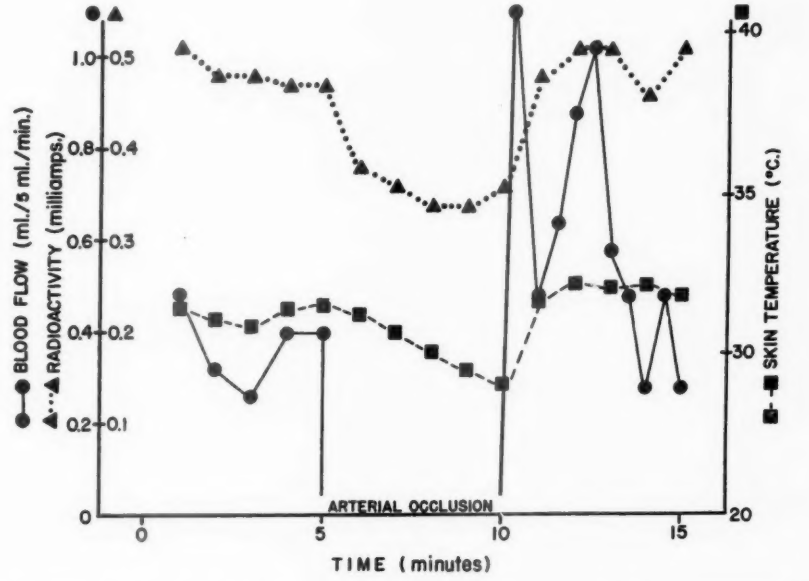

Fig. 10.—The points on the three graphs represent averages of determinations made during one-minute periods as in Fig. 9, except that the hand was in a state of vasodilation and not of vasoconstriction, as it was in Fig. 9. Note the prompt responses of the rate of blood flow and of the level of radioactivity, but the slower response of skin temperature.

above the preocclusion level. Skin temperature in the vasoconstricted hand reaches a level decidedly above the control, but at a slower rate than either blood flow or radioactivity.

In both the vasoconstricted and vasodilated hands, the rate of blood flow returns to nearly normal as early as five minutes after the release of the tourni-

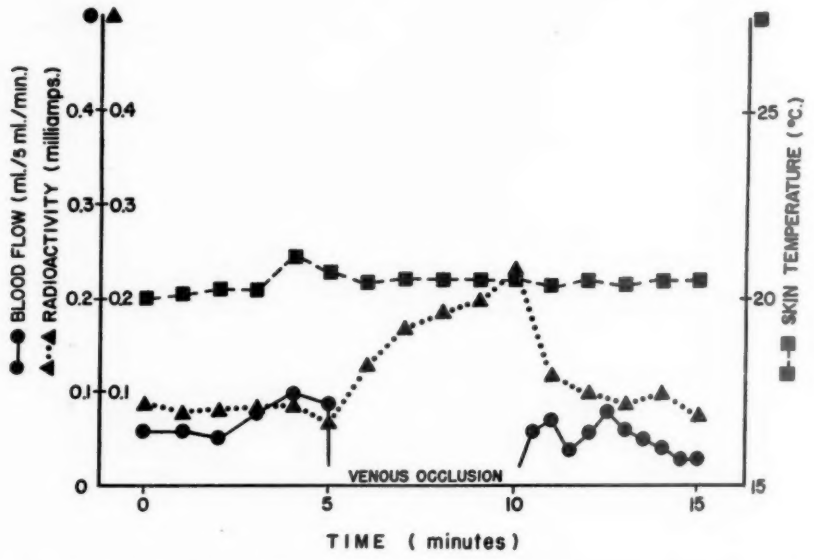

Fig. 11.—The three graphs illustrate the responses to venous occlusion. Between five and ten minutes on the abscissa, the sphygmomanometer cuff on the arm was inflated to 70 mm. Hg. Note the prompt decline in radioactivity with release of the occlusion. The hand was initially vasoconstricted with the room temperature constantly at 20° C.

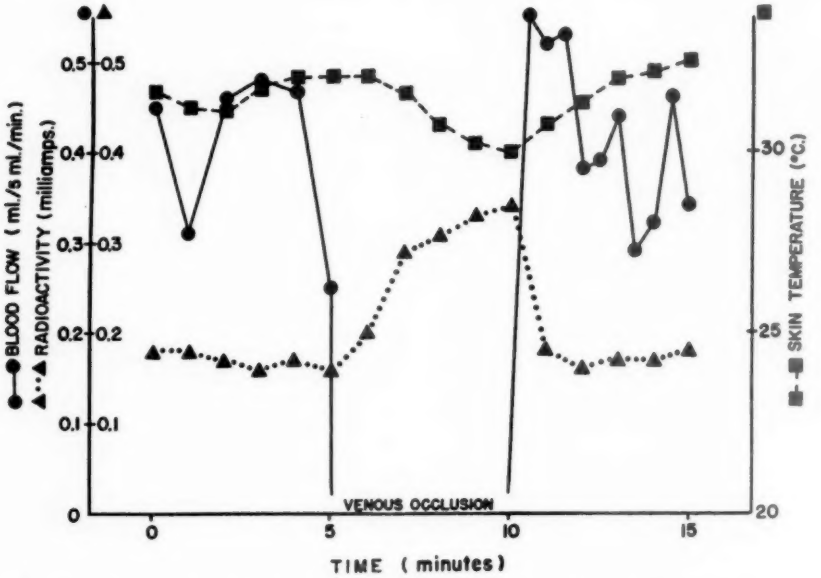

Fig. 12.—These graphs are similar to those of Fig. 11, except that the hand was initially vasodilated. Note the prompt decline in radioactivity upon release of the venous occlusion. There was no evidence of diffusion of the radioactive albumin into the extravascular tissues.

quet. Skin temperature does not fall as long as reflex vasodilation persists; however, in the previously vasoconstricted hand and with the ambient temperature constant at 20° C. the temperature of the skin returns very slowly toward the preocclusion level.

In the previously vasodilated hand, radioactivity remains at approximately the control level after release of the arterial occlusion. In the vasoconstricted hand, radioactivity falls toward normal after its postrelease maximum, but it does so slowly. In contrast, accumulated radioactivity always declines very rapidly following release of venous occlusion (Figs. 11 and 12).

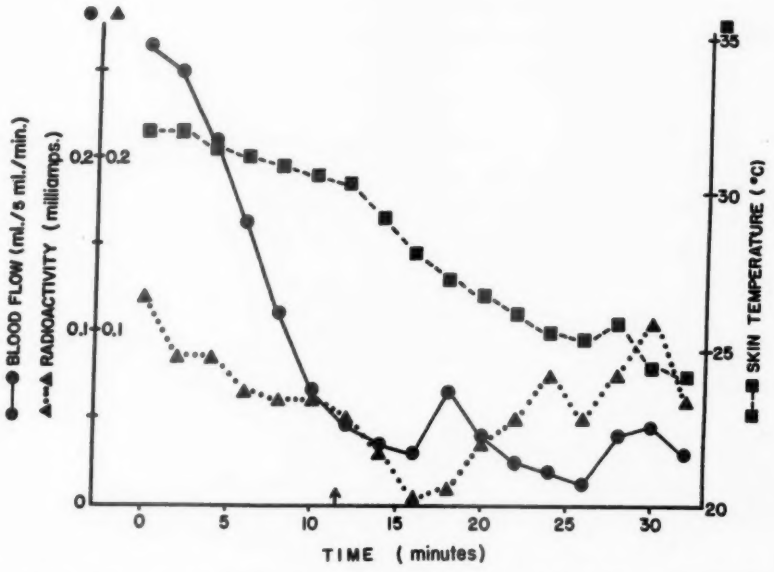

Fig. 13.—The three graphs describe the changes of blood flow, radioactivity, and skin temperature of the finger in response to reflex vasoconstriction. At the arrow, legs and feet were immersed in water at 10° C. and remained so for the duration of the experiment. Note that the curves indicate vasoconstriction even before immersion of the legs. This is attributed to the subject's awareness of the preparations for the test which were begun at zero time. Vasoconstriction is demonstrated by all three methods, but more slowly by skin temperature change than by changes of the other two indices. There is a gradual rise in radioactivity toward the latter part of the experiment despite the continued low rate of blood flow as indicated by the other two graphs (see text).

Rate of Response to Reflex Vasoconstriction and Vasodilation.—Fig. 13 demonstrates the effect of immersion of the feet and legs in water at 10° C. Preparation of the water bath started at zero time was obvious to the subject and caused a fall of all the entities under study, a fall which was accentuated by the actual immersion, indicated by an arrow on the graph. A tendency for the radioactivity to increase unaccompanied by a rise of blood flow and skin temperature is present in the latter period of the immersion shown on the graph. This may mean that part of the initial fall in the level of radioactivity was due to a venular constriction which later relaxed despite persistence of low blood flow and skin temperature. No example of this phenomenon was seen in any other patient, however. The data again show that the radioactive material is essentially confined to the functioning blood vessels and that measurement of

blood flow and of radioactivity are more sensitive indices than changes in skin temperature as measured here, in that they respond more quickly to changes in the circulation.

The converse situation of immersion of the legs and feet in water at 45° C. when vasoconstriction was present is depicted in Fig. 14. The chart shows the expected rise with all three variants, the rate of rise of blood flow and radioactivity preceding the change in skin temperature. Very low orders of radioactivity resulted from the dosage of I^{131}-labeled albumin used here. This made it necessary to obtain an Esterline-Angus record for at least thirty seconds before an upward or downward trend in the level of radioactivity could be recognized as significant. When rapid fluctuations in blood flow were associated with alterations in digital blood volume, such alterations, as measured by their radioactivity, were indicated by smoothed rises or falls in the milliammeter record.

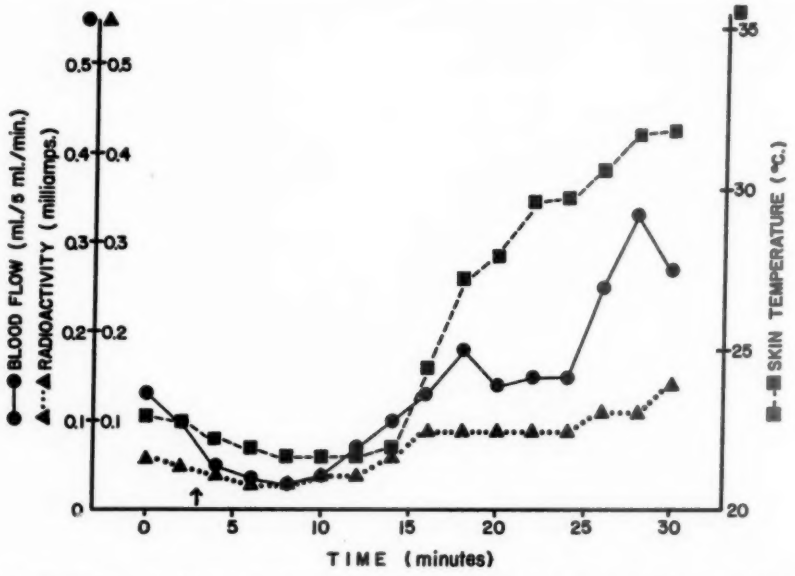

Fig. 14.—Changes in blood flow, radioactivity, and skin temperature of the finger in response to reflex vasodilation are shown here. At the arrow, the legs and feet were immersed in water at 45° C. and remained so for the duration of the experiment. Ambient temperature was 20° C. Vasodilation is indicated by all three methods of study, but slightly more slowly by the skin temperature changes than by those of the other two.

Examples of this response were repeatedly seen during the course of arterial occlusion and release studies. Individual occlusive plethysmographic cuff inflations were not constantly associated with rises of recordable digital radioactivity; however, a series of such inflations spaced at short intervals (for example, ten seconds apart) caused a rise of the Esterline-Angus tracing during this period. Despite this relatively slow response, rise or fall of blood flow and radioactivity always preceded any alteration in skin temperature.

DISCUSSION

The three techniques studied here could not be expected to parallel exactly each other's rise and fall as they are not dependent upon alterations of the same variable. As determined here, the rate of blood flow is a measurement of

the rate at which blood accumulates within the digit when the venous outflow from the digit is occluded. The level of radioactivity, on the other hand, reflects the amount of blood in the finger at any given moment. It represents a measurement of rate of blood flow only to the point where all of the available vessels are filled with radioactive material. Beyond this point, the intensity of radioactivity will not change with variations in the rate at which that blood is passing through the finger. Instead, it will change with variations in the volume of blood contained within the digit at any one time. With the amounts of radioactive material used here, it is impractical to use this technique for plethysmography. Skin temperature, as measured here, is the resultant of ambient temperature and the temperature imparted to the skin by the blood circulating through it. It can describe changes of the rate of blood flow through the skin provided there is a sufficient gradient between the temperature of the blood and that of the environment.

Plethysmographic blood flow recordings in a finger or toe are generally satisfactory until high rates of flow occur. Rapid filling of the capacity vessels then distorts the slope[7] of the tracing obtained and makes calculation of the rate of flow less certain than when two or more waves are necessary to fill the digit. Moreover, cuff artifacts may give rise[8] to difficulty in the calculation of the data. The necessary apparatus for plethysmography is generally more complex than that used for skin temperature determinations. Nevertheless, the method has been widely used and has given satisfactory results subject to these limitations.

Measurement of circulating radioactivity also requires a complex array of apparatus. As has been previously described, the technique used here has provided a satisfactory working margin (50 per cent) of total recordable radioactivity. The rate of rise and fall of radioactivity is slow and a period of over thirty seconds has been required to decide whether a trend in the tracing is likely to have significance. However, the changes are faster than those of the temperature of the skin as measured by the means used here, and they are definite, provided that the finger has been kept still in the counting chamber. Artifacts are liable to occur when the patient becomes restless toward the end of a lengthy study.

The temperature of the digital skin depends upon the amount of heat brought to it by blood flow in the subcutaneous vessels and the amount lost to the environment. It is unlikely that temperature will fall below that of the environment unless much heat is lost by evaporation of sweat. It is also unlikely that it will rise much above 35° C. unless the patient becomes febrile or environmental temperature passes this figure. Hence, the maximum range of temperature available in any study is the difference between that of the environment and somewhat below that of the body. Montgomery and his colleagues[9] have presented graphs of digital skin temperatures plotted in relationship to blood flow at various environmental temperatures. These graphs are very similar to those shown in Figs. 7 and 8, although higher flow rates were required by Montgomery's subjects to raise their skin temperatures to levels comparable with those of our patients. Nevertheless, it appears from our results that when

digital blood flow is greater than 1 ml. per 5 ml. of part per minute with an environmental temperature of 20° C., skin temperature is not likely to rise further with increase of blood flow. Indeed, the temperature of the skin may not rise beyond that representing "maximal vasodilation" despite the fact that the rate of blood flow may increase to five times that required to raise skin temperature to its "maximal" level.[10] With lower ambient temperatures as suggested by Fetcher and his colleagues,[11] close correlation between skin temperature and blood flow over a broader range of blood flows is possible. Such very low temperatures, however, are not practicable for use with patients in routine clinical investigation, and they would have a direct effect per se on digital blood flow.[12]

When the records of the three variants studied and presented here are compared, it can be seen that individual rises and falls under the varying conditions occur much as would be expected. There are, however, certain interesting exceptions. In Figs. 2 and 9, arterial occlusion in the vasoconstricted hand has resulted in a reactive hyperemia accompanied by a rise of skin temperature and circulating radioactivity above the initial control levels. When, however, the hand was first vasodilated (Fig. 10), no corresponding rise of skin temperature and radioactivity usually occurred with the additional rise in blood flow. The explanation of this phenomenon may lie in the redistribution of flow pattern during the phase of reactive hyperemia. Thus, if arteriovenous shunts opened and short-circuited the relevant capillary bed, it is possible to conceive that flow could rise in the absence of an increase of digital blood volume.

Rerouting of blood through capillaries with closure of arteriovenous communications may also explain the rise of circulating radioactivity in the absence of a corresponding rise in blood flow already noted in Fig. 13. It should prove interesting to extend this study to sympathectomized subjects and discover whether this observation can be repeated in digits with no vasomotor control.

The radioactive isotope methods used by Smith and Quimby,[13] by Krieger and his colleagues,[14] and by Harris and his colleagues[3] are essentially different from the technique used here. These workers obtained information regarding rates of blood flow by measuring the rate of accumulation of radioactive sodium or of I^{131}-labeled albumin or by measuring the rate of clearance of radioactive sodium injected extravascularly. The technique used here gives no such information, but indicates only the variations in blood volume in the part under study, variations which are not always capable of simple interpretation. Rates of build-up and of clearances of radioactive substances could be adapted for the study of digital blood flow in the hand or foot, but they would not give more information than the technique described in this report or than could be obtained by digital plethysmographic methods. Moreover, the response of the vascular bed to such stimuli as arterial and venous occlusion could hardly be studied by the build-up methods.

The applicability of observations on skin temperature to studies of the peripheral circulation is limited by the relationship between body temperature and ambient temperature and by the loss of relationship between skin tempera-

ture and blood flow at high rates of flow. For the routine clinical assessment of the peripheral circulation, however, observation of changes in the temperature of the skin with controlled environmental temperature under different test situations is generally adequate. For the detailed investigation of specific effects of clinical therapeutic measures, plethysmography or isotope techniques can provide additional information; of the two, plethysmography is the easier.

SUMMARY AND CONCLUSIONS

1. A comparison of blood flow, circulating radioactivity after an intravenous injection of I^{131}-labeled serum albumin, and skin temperature has been made in the finger.

2. There was no evidence that the isotopic tracer material escaped into the extravascular tissue spaces in amounts sufficient to interfere with the tests.

3. Radioactive isotope techniques can provide useful information regarding the peripheral circulation, but they are cumbersome and have other limitations.

4. Provided its limitations are appreciated, measurement of skin temperature is a useful and reliable method for assessing the efficiency of the digital circulation for the usual clinical purposes.

5. For more precise information, estimates of digital blood flow by means of plethysmography can provide useful data beyond those that can be obtained from studies of the temperature of the skin.

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OCCLUSION PATTERNS AND COLLATERALS IN ARTERIOSCLEROSIS OF THE LOWER AORTA AND ILIAC ARTERIES

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INTRODUCTION

OBSERVATIONS bearing on the pathology of arteriosclerosis of the lower aorta and the iliac arteries have been scanty as regards the sites of obstruction and the collateral pathways around the obstructions. The present report deals with 37 aortograms scrutinized to determine the pattern of arteriosclerotic involvement and the identity of the collaterals. The anatomic data are also briefly correlated with the clinical adequacy of the circulation.

As an ancillary study, stereoscopic roentgenography and dissection were carried out on cadavers whose arterial systems had been injected with an opaque medium, as an aid in identifying the branches of the hypogastric artery in the living.

In the description of each collateral pathway which is to be presented, a distinction will be made between the artery originating above the obstruction and carrying blood distally, and the artery recurrent in direction, which receives this blood to conduct it to the arteries below. We designate the proximal vessel the "affluent" of the collateral pathway, the distal one the "effluent." In some instances, obstruction of lower vessels makes a secondary or "intermediate" anastomosis necessary before the blood can reach its destination.

The change from the single large, normal artery to the multiple smaller collaterals always involves some diminution in blood flow, since according to the law of Poiseuille, the quantity of liquid flowing through a tube is equivalent to the fourth power of its radius. The degree of this diminution in flow is thus directly dependent upon the width of the arteries in the collateral pathway. When the vessels are narrow, the length of the path is likewise important, resistance to flow being proportionate to the length of vessel involved.

Vessel width is particularly significant with regard to the mode of continuity of the affluent and effluent channels, a bold connection of the two (inosculation) offering no additional resistance to flow, while a connection via fine branches (retiform anastomosis) introduces additional drops in pressure and volume flow. The size of anastomotic channels was considered in its

Aided by a grant from the Massachusetts Heart Association.

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

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historical perspective by Olovson.[1] He pointed out that as anatomists of the eighteenth century discovered new collaterals, these were overemphasized in illustrations to create an impression of gross continuity of anastomosing arteries. Only recently has it been emphasized that the connections are usually small, and the anastomoses sometimes inadequate.

MATERIAL

The cases studied were derived from the Peter Bent Brigham, the West Roxbury Veterans Administration, and the Boston Veterans Administration Hospitals.*

A total of 37 aortograms was selected from 50 consecutive cases, the remainder being discarded either because the essential problem was not obstruction or because the films were not sufficiently clear. Of the 37, 4 were women. Six patients were diabetic; only 1 of these was a woman. The patients have been observed from one to several years.

In about half the cases, multiple films showing different phases of the injection were available. Films of the femoral arteries had been made in most instances, and the arteries of the popliteal region had been visualized in many.

Representative films are presented side-by-side with labeled tracings of the arteries.†

ANALYSIS OF THE AORTOGRAMS

General Pattern of Occlusion.—The highest level of major obstruction, defined as 75 per cent to total, is given in Table I. There was little tendency to collateral formation until the obstruction was close to total or total.

TABLE I. HIGHEST LEVEL OF MAJOR OBSTRUCTION

	CASES	CLINICAL COMMENT	
		DIABETICS	THIGH AMPUTATION
Aorta	6	0	1
Common iliac	23 (inc. 2 bilateral)	5	3 (inc. 1 bilateral)
External iliac	8	1	3
Total	37	6	7

TABLE II. ASSOCIATED OBSTRUCTIONS

	LEVEL OF HIGHEST MAJOR OBSTRUCTION			TOTAL CASES WITH LACK OF DEMONSTRATION (PER CENT)
	AORTA	COMMON ILIAC	EXT. ILIAC	
Inferior mesenteric artery				
Potentially visible*	6	16	5	
Not demonstrated	3	6	1	37
Internal spermatic or ovarian artery				
Potentially visible*	4	8	1	
One or both not demonstrated	4	5 (Bilat. 3)	1 (Bilat.)	76 (one or both arteries)
Hypogastric artery				
Potentially visible†	5	22	8	
One or both not demonstrated	1	6 (Bilat. 3)	3 (Bilat. 1)	29 (one or both arteries)

*Injection at adequately high level.
†Adequate demonstration of other vessels in pelvis.

Table II lists the incidence of failure of filling of three significant branches of the main trunk. When the injection had been made at a level adequate

*We want to express our thanks for cooperation in this study to Drs. Richard Warren, Henry Faxon, Merrill Sosman, and Chilton Crane.
†We are grateful to the artist, Miss Mildred Codding, for these drawings.

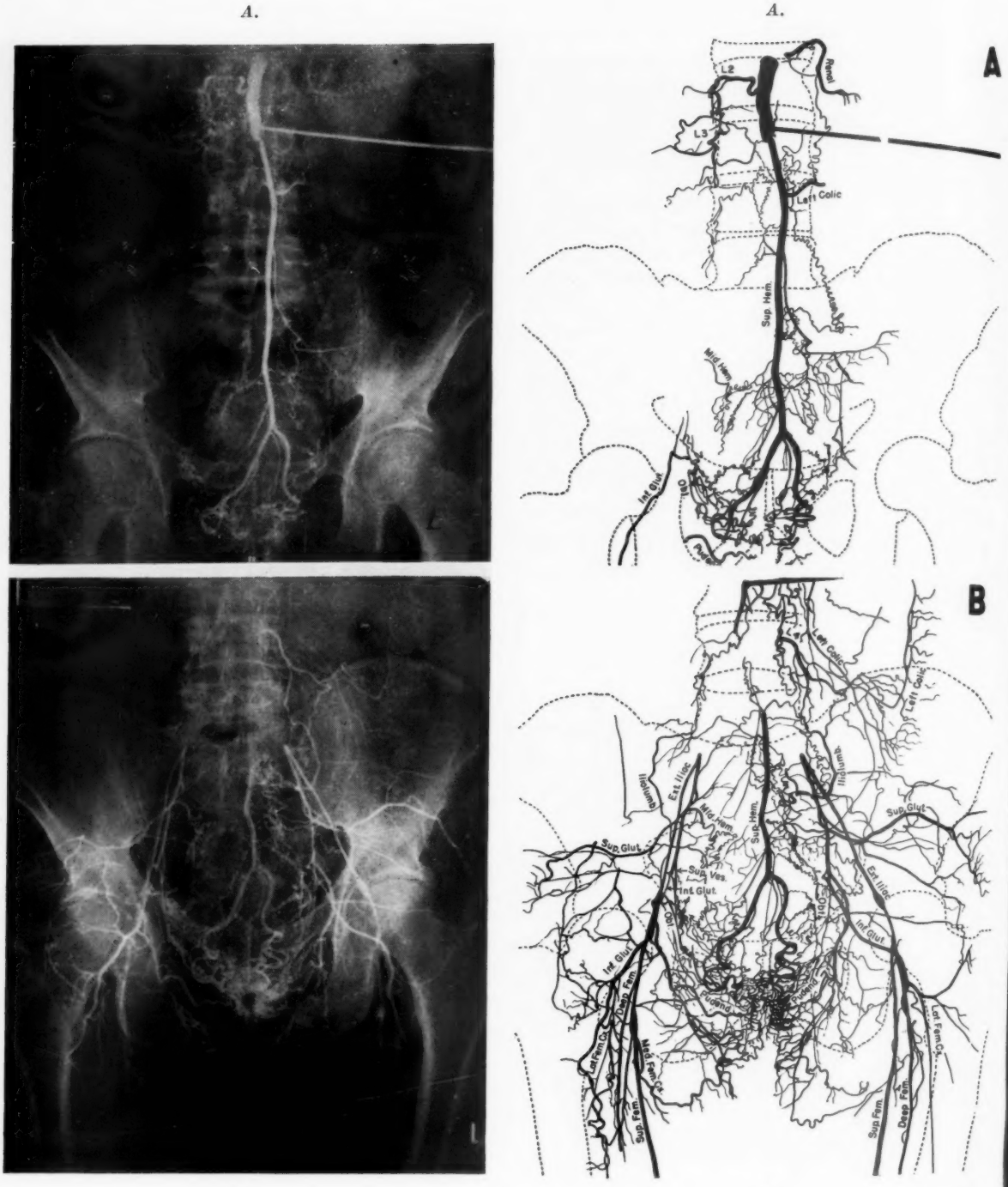

Fig. 1.—Aortic obstruction. *A* and *B* are two phases of a single injection. In *A*, the rectal vessels are beginning to fill via the inferior mesenteric artery. In *B*, the medium has extended to an excellent iliac and femoral system via numerous fine mid- and inferior hemorrhoidal vessels. This patient suffered severe claudication and neuritis, relieved by endarterectomy.

to show the inferior mesenteric artery, this vessel failed to be filled with the opaque medium in 37 per cent. Similarly, the internal spermatic or ovarian arteries were not demonstrated on one or both sides in 76 per cent. One or both hypogastric trunks failed to fill in 29 per cent of films in which other pelvic arteries were well demonstrated. Under the circumstances mentioned, it is reasonable to believe that most, if not all, of the vessels thus unfilled were occluded.

The condition of the femoral system, including the superficial and deep femoral arteries, will be considered separately for each level of obstruction.

Cases With Aortic Obstruction.—

A. *General data*: Six patients were in this category; 1 was a woman; none was diabetic. All 6 showed total obstruction of the aorta at some level below the renal arteries. As Table II shows, there was frequent occlusion of the internal spermatic and inferior mesenteric arteries. The aortic occlusions were usually distinguished, however, by freedom of occlusion of the iliac and femoral systems (Fig. 1). The hypogastric trunks were occluded in but 1 patient; in another these were patent, but there was occlusion of the common and external iliacs and the femoral arteries on both sides (Fig. 2).

The femoral systems were excellent in 4 patients. There was one thigh amputation in the entire group of 6 patients, associated with extensive occlusion of the femoral and deep femoral arteries. Function in the remaining 5 patients varied considerably, with and without treatment, but massive necrosis was not a problem in them.

B. *Collaterals*:

1. Rectal. These collaterals were the most consistently used. The superior hemorrhoidal was the major affluent (Fig. 1). When the origin of the inferior mesenteric was occluded, this artery was filled via the "marginal artery" of the colon, bringing blood from the middle colic branch of the superior mesenteric to the left colic, and thus to the lower part of the inferior mesenteric (Fig. 2). The effluents of the rectal pathway were exceedingly numerous and were usually fine vessels which extended above the pelvic diaphragm to every branch of the hypogastric except the iliolumbar. Other visceral branches of the hypogastric, such as the vesical, were probably used also, but we found their identity hard to establish. The cadaver study also indicates that large branches to the levator ani muscle, often arising from accessory pudendal arteries, may act as effluents. Below the pelvic diaphragm, effluent channels were the inferior hemorrhoidal branches of the internal pudendal, and occasionally of the obturator, external pudendal, and medial femoral circumflex arteries. The intrapelvic effluents carried the medium back to the hypogastric arteries and thus to the common and external iliac arteries. When the common iliac was blocked, the hypogastric or its branches acted as intermediate vessels for flow to the femoral system via a second collateral junction (Fig. 2).

2. Iliac. An anastomotic pathway was established in most cases along the iliac crest. The affluents here were usually the second, third, and fourth

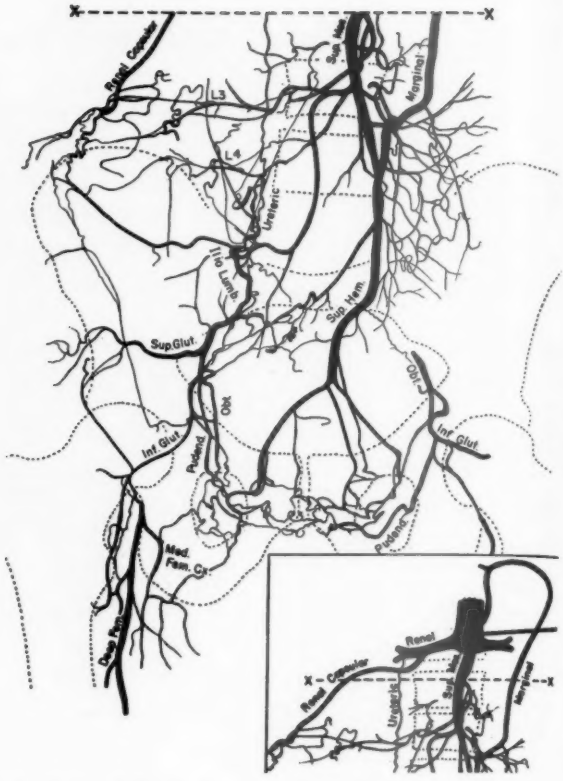

Fig. 2.—Aortic obstruction with occlusion of origin of the inferior mesenteric. The rectal system is filled from the superior mesenteric through the "marginal" artery (between the mid- and left colic). Iliac anastomoses are also present. There is concomitant occlusion of the common and external iliac arteries. The right hypogastric creates intermediate anastomoses in the path to the limb. Fair function without treatment.

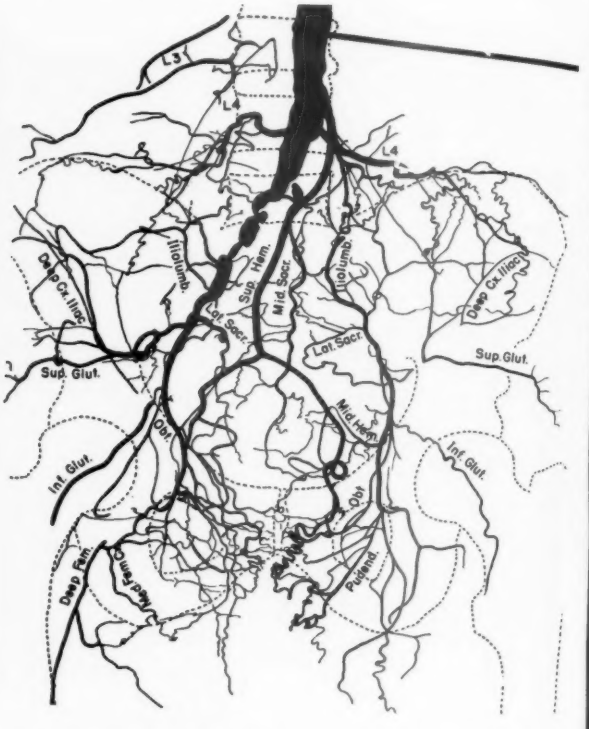

Fig. 3.—Bilateral common iliac obstruction extending to the external iliac and femoral arteries. Major collaterals are the rectal and iliac. The loss of both limbs in this patient correlates with the extremely diffuse involvement.

lumbar arteries. One or more of these vessels was often occluded, leaving a single one as a bold contributor to the anastomosis. In three cases a large adipose capsular branch of the renal artery also led to the anastomosis. The effluents anteriorly were the iliolumbar and deep circumflex iliac arteries, posteriorly and less striking, the superior gluteal. Again, a secondary set of anastomoses might be interposed in the route to the limb when the iliac system was imperfect.

3. Other collaterals. A comment may be made on these. Because its origin was occluded, the mid-sacral artery could not act as an affluent, but portions of the artery sometimes acted as an intermediary for neighboring vessels.

Our injections, not being made in the arch of the aorta, could not have filled the internal mammary-inferior epigastric pathway. It is nevertheless unlikely that this anastomosis was of great significance in our cases, since the medium in the external iliac arteries did not seem diluted by blood near the inferior epigastric origin, and, indeed, in some instances, the inferior epigastric was well filled by the contrast medium from below.

The injection was again usually too low to show whether lower intercostal vessels take part in the iliac anastomosis. From operative and anatomic evidence, it appears that this pathway may be of occasional importance and we have seen one aortogram, with occlusion at the juxtarenal level, in which the left eleventh intercostal artery was a major affluent.

Occasionally the ureteric vessels were prominent (Fig. 7), but they were never large and appeared to be unimportant as collaterals. The frequent occlusion of the spermatic arteries has been noted. When patent, the vessels appeared to send no branches to any structure beyond the testes.

Cases With Common Iliac Obstruction.—

A. *General data*: There were 23 cases in this category, in 2 of which both common iliacs were obstructed. In the remainder, the artery was occluded 10 times on the right side, 11 times on the left. Three patients were women; one of these was included in the 5 diabetic patients in the group.

The common iliac cases showed a strong tendency toward diffuseness of the arteriosclerotic process. Partial occlusion involved almost every contralateral common iliac as well as the external iliac and femoral arteries of each side. Concomitant total occlusion of the external iliac, often extending to the common and superficial femoral arteries, occurred on the same side as the common iliac occlusion in 3 cases, on the opposite side twice, and in both external iliacs once. Table II shows the high incidence of obstruction of the inferior mesenteric, sex, and hypogastric arteries also noted in the common iliac group.

One of the patients with bilateral common iliac occlusions came to bilateral thigh amputation, and a thigh amputation was also necessary in each of 3 patients with obstruction of a single common iliac. Amputations were usually associated with extremely diffuse occlusions. Two of the 4 amputees were diabetic.

B. *Collaterals:* The affluents of the anastomotic channels in the bilateral occlusions were similar to those seen in the aortic obstructions. Theoretically, the mid-sacral artery can aid in the case of iliac obstructions, but its origin was occluded in both instances. The striking difference in the anastomoses for the two levels of obstruction was the more frequent interposition of intermediate vessels in the effluents in the case of the iliac obstructions, necessitated by the more diffuse arteriosclerotic process (Fig. 3).

In both the bilateral and unilateral occlusions, the effluents often had to by-pass an obstruction of the external iliac as well as of the common iliac. For the sake of simplicity, the following classification of collaterals in instances of unilateral common iliac obstruction assumes the patency of the external iliac artery.

1. Iliac. This was by far the most important pathway and was constituted by essentially the same vessels as in aortic obstruction except that the affluents came from a lower level. Thus, of the lumbar arteries, only the fourth took part (Fig. 4); no renal contributions were seen. Again, the intercostal arteries were not observed to take part in the anastomosis.

2. Sacral. In this anastomosis between the mid- and lateral sacrals, the mid-sacral was sometimes the major affluent (Fig. 5); in other cases it was the opposite lateral sacral. The vessels of the pathway were usually narrow. Rectal arteries sometimes aided as intermediates in paths to other branches of the hypogastric.

3. Transverse. These were numerous but generally fine vessels extending from branches of the opposite hypogastric and femoral arteries to corresponding branches of the affected side (Fig. 6). Posteriorly these included the lateral sacrals; anteriorly, within the pelvis, the various visceral branches of the hypogastric (usually difficult to identify with certainty), somewhat larger connections between the obturators and internal pudendals; and finally, the medial femoral circumflex and external pudendal branches of the femoral arteries.

4. Rectal. These anastomoses did not seem to come into play unless there was extremely diffuse involvement. Compare, for example, the undilated superior hemorrhoidal artery in Fig. 5 with the large vessel in Fig. 3.

Cases With External Iliac Obstruction.—

A. *General data*: As mentioned, in several instances complete obstruction at this level complicated common iliac obstruction. There were 8 instances, in addition, in which the highest major occlusion was located in a single external iliac artery. Four of these were on the right, 4 on the left. The patients were all men. Thigh amputations were performed in the one diabetic and the 2 other patients of this series.

Patients with external iliac occlusion showed the same tendency to diffuse involvement of the arteries already noted for the common iliac occlusions (Table II). Proximally the obstruction usually started immediately below the termination of the common iliac. Distally, the entire external iliac was usually involved, and often the common femoral artery was also occluded. Further occlusion of the superficial femoral was common.

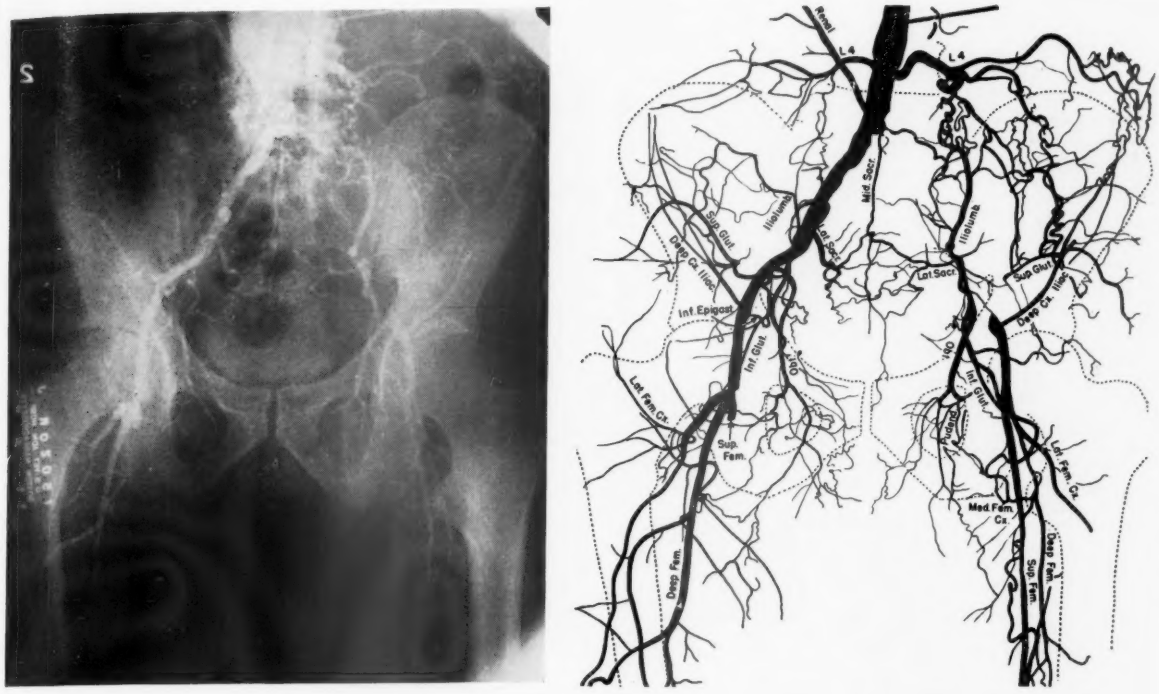

Fig. 4.—Obstruction of the left common and external iliac arteries. The iliac collaterals extend partly to the intermediate anastomoses of hypogastric branches and partly to the deep circumflex iliac. Both superficial femoral arteries are occluded. A rare accessory right renal artery is seen. Fair function without treatment.

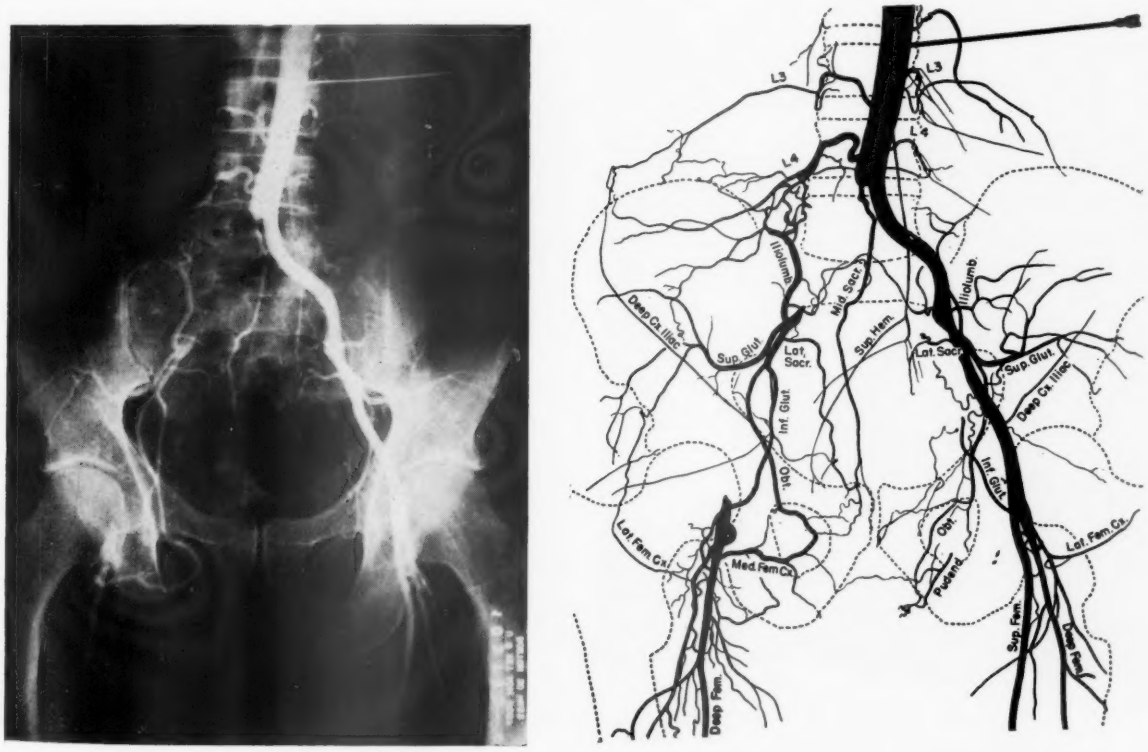

Fig. 5.—Obstruction of the right common and external iliac arteries, extending to the common and superficial femorals. The inferior mesenteric is visualized but takes no part in the anastomoses. The iliac collaterals are the major ones, the hypogastric artery acting as intermediary; the sacrals assist. An obturator-medial femoral circumflex inosculation is evident; the superior and inferior gluteals form fine anastomoses; the internal pudendal does not fill. This limb came to amputation.

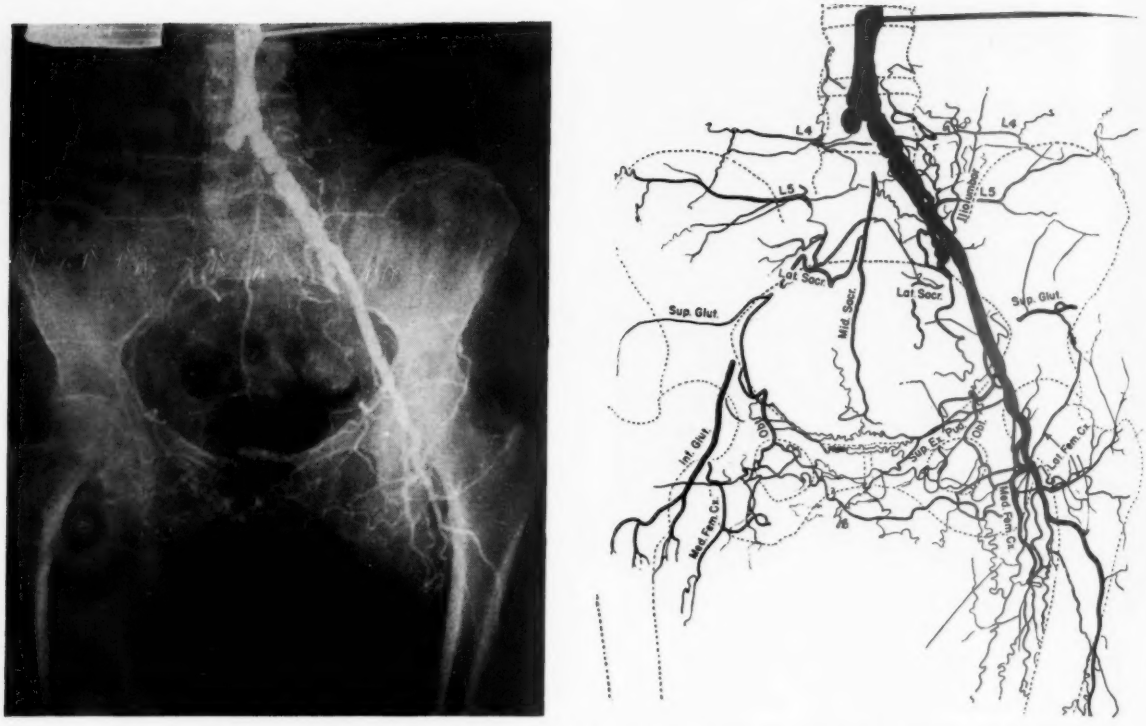

Fig. 6.—Unusually diffuse obstruction of all major trunks of the right side. The major anastomoses are transverse. The right limb was lost; the left is in good condition after sympathectomy, in spite of occlusion of the superficial and deep femoral arteries.

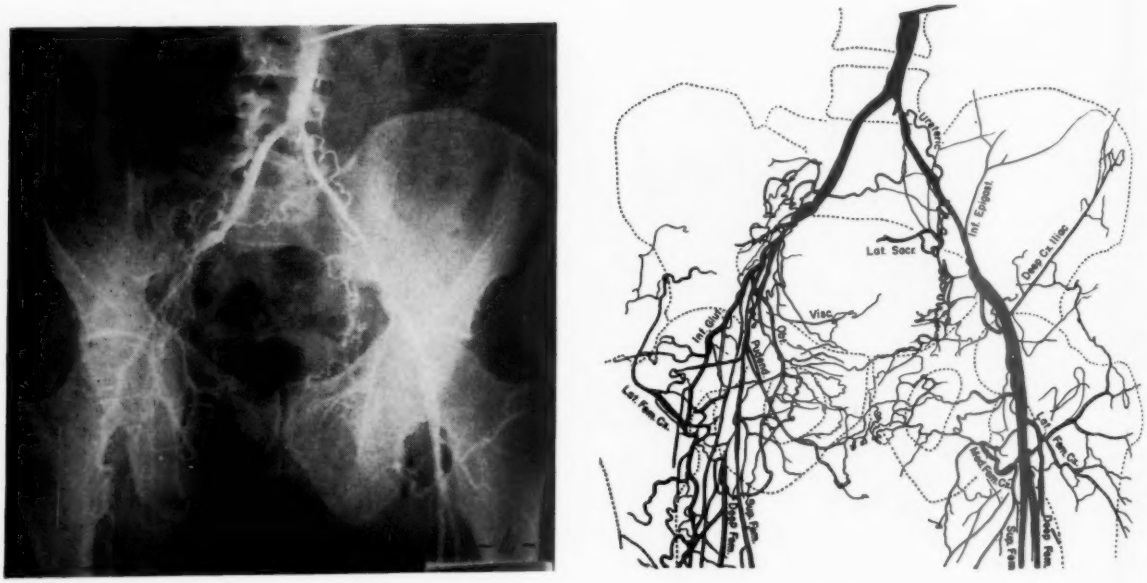

Fig. 7.—Obstruction of the right external iliac artery is bridged by descending hypogastric collaterals. On the left side common iliac occlusion, short of total, is unaccompanied by collateral formation. Fair function after sympathectomy.

B. *Collaterals*: 1. Descending hypogastric. The major affluents were the medial descending branches of the hypogastric, the obturator, the inferior gluteal, and, to a much smaller degree, the internal pudendal. The medial femoral circumflex was the main effluent, and its connection with the obturator often constituted an inosculation and was the best example of this type of anastomosis seen anywhere in the aortograms. The inferior gluteal was also prominent, but its terminations were much more tenuous, its anastomosis, with fine branches of the profunda femoris and medial femoral circumflex, difficult to find. The branch of the inferior gluteal to the sciatic nerve was rarely identified. The internal pudendal artery was often occluded; when patent, it sent fine branches to join the medial femoral circumflex (Fig. 7).

One lateral descending branch, the superior gluteal, occasionally connected with the ascending branch of the lateral femoral circumflex, but this connection was fine and inconstant (Fig. 5).

2. Rectal. This pathway was quite unimportant except in the presence of very diffuse disease (Fig. 3).

3. Iliac. A connection between the fourth lumbar artery and the deep circumflex iliac was significant in the one instance in which the iliac obstruction stopped short of the terminal part of the external iliac artery (Fig. 4). The iliolumbar probably shares in a path to the deep circumflex iliac in similar cases where the common iliac is intact.

4. Collaterals in combined common and external iliac obstructions. Significant channels included the rectal (Figs. 2 and 3), the iliac (Figs. 4 and 5), mid-sacral (Fig. 5), and transverse anastomoses (Fig. 6), Intermediate anastomoses were often in use in these pathways.

COMMENT

1. *Consequences of the Occlusion Pattern.*—Patients with total occlusion of the lower aorta appear to belong to a group apart from those with occlusion of the iliac levels. As a rule, aortic occlusion occurs in somewhat younger patients, diabetes is unusual, and the process is more localized than when occlusion begins at a lower level. Thus, while aortic occlusion is disabling, gangrene is unusual, a fact almost universally commented on in references to this lesion.

The occurrence of gangrene, therefore, does not appear to be related to the height of occlusion or to the particular collaterals used. It is related occasionally to arteriosclerotic involvement of collateral channels, but more particularly to diffuse involvement of the ultimate distributing vessels. It should be noted, however, that the occurrence of gangrene often bears no particular relation to the variety of anatomic lesion, but is rather dependent upon external incidents such as injury or infection.[2]

Mention has been made that blood flow through collaterals rarely equals that through the major arteries normally supplying an area. Thus, not only does the limb suffer from occlusion of the aorta and iliac arteries, but we may also expect some ischemic symptoms in structures dependent upon the frequently occluded inferior mesenteric, spermatic, or ovarian, and hypo-

gastric arteries. It may well be that some of the digestive disturbances of old age depend upon obstruction of the inferior mesenteric, and that diminution of testicular and ovarian activity has a similar basis. Incidentally, we were unable to demonstrate with certainty the collaterals replacing the occluded internal spermatic which have been described by Neuhof and Mencher.[3]

Obstruction of the hypogastric artery has the obvious effect of producing atrophy and loss of power of the gluteal musculature, the deficiency in blood flow being compensated for but poorly.[4] Branches of the hypogastric also supply the large lumbosacral nerve plexus and its continuation, the sciatic. It is known that ischemic neuritis develops easily in nerves whose nutrient vessels are compromised and there is suggestive evidence that neuritis of the sciatic nerve may occur on such a basis.[4] The demonstration of frequent hypogastric obstruction strengthens the probability that ischemia is responsible for some instances of extensive neuritis in arteriosclerotic patients, particularly those with diabetes, especially since it is known that arteriosclerosis of pelvic arteries is so frequent in that disease.[5]

2. *Identity of the Collaterals.*—The present account of the collaterals is perhaps a more systematic description of the situation in arteriosclerosis than has heretofore been attempted, but various pathways have been mentioned by many writers, beginning in the eighteenth century. The reports of Olovson[1] and of Burt, Learmonth, and Richards[6] are particularly comprehensive. Certain anastomoses may be commented on here. The importance of the rectal collaterals was noted by Chiene[7] in 1869 in a patient with mesenteric obstruction, and he postulated that this might be the most significant pathway to the limbs in aortic obstruction. We agree with Lindström[8] that the inferior mesenteric is often the decisive collateral in obstruction at this level. In the patient whose films are shown in Fig. 1, temporary occlusion of the inferior mesenteric during operation resulted in a slight but definite disturbance in the calf muscles. Baylin[9] reported a patient in whom the inferior mesenteric was filled by the anastomosis between the mid-colic and left colic, which was also prominent in two of our patients (Fig. 2). The normal constancy of this anastomotic connection has been documented by Steward and Rankin.[10] It appears quite certain that if all our injections had been made at a level adequate to fill the superior mesenteric, this pathway would have been demonstrated in the majority of patients with occlusion of the origin of the inferior mesenteric. The normal richness of the anastomoses of the rectal arteries with branches of all the neighboring pelvic arteries has been demonstrated by Pope and Judd.[11, 12]

The possible role of the internal mammary-inferior epigastric path remains unsettled. This is known to be an important channel in patients with coarctation of the aorta[13]; yet it is frequently insignificant even in that condition.[14] We may re-emphasize that the inferior epigastric filled from below in some of our films, indicating that, in these cases at least, the artery did not constitute part of a collateral pathway.

3. *Size and Adequacy of the Anastomoses.*—That the anastomotic connections between vessels in the collateral pathways to the lower limb are usually fine was mentioned by many nineteenth century writers[1] and has recently been emphasized by Rassat[15] and Olovson[1] for the collaterals about experimental obstructions of the external iliac artery. It will be noted that we found very few large connections between the affluent and effluent arteries, the only frequent examples of an inosculation being the junction of the obturator and medial femoral circumflex arteries. Occasionally, the union of the fifth lumbar and iliolumbar arteries was also of this order. The anastomosis between the inferior gluteal and the vessels of the thigh was disappointing, in view of the substantial diameter of the inferior gluteal itself, and the embryologic position of that vessel as the axial artery of the limb. As a rare anomaly, the inferior gluteal may constitute the major artery to the limb and it therefore seems probable that, in a large series, this artery may occasionally be seen forming an inosculation with the profunda artery or one of its branches.

The significance of the size of the anastomotic channels has been summed up by Leriche, "One cannot count on macroscopic anastomoses to assure, under all circumstances, the maintenance of a sufficient circulation in a limb in case of (operative) obliteration of the principal pathway."[1]

4. *Injury to the Collaterals.*—It appears that since the circulatory pathway to the limbs may depend upon a single vessel, such as the superior hemorrhoidal or fourth lumbar artery, operative damage to such a vessel may cause the death of one or both limbs. This may explain some instances of gangrene after operations in the abdomen or pelvis. Several situations in which such an injury could occur come to mind: the removal of the rectum for carcinoma, the division of a lumbar artery by a flank incision carried far posteriorly, or the accidental damage of the obturator artery in an operation for hernia or in obturator neurectomy.

SUMMARY

An analysis of 37 aortograms showing arteriosclerotic occlusion of the lower aorta or iliac arteries is presented as a basis for determining the pattern of occlusion, and the collateral channels used in this disease. Considering those films made with adequately high injection, the inferior mesenteric artery failed to fill in 37 per cent, the internal spermatic or ovarian in 76 per cent (on one or both sides), and the hypogastric in 29 per cent (on one or both sides).

Obstruction at the aortic level, which occurred in 6 cases, tended to be more localized than when obstruction began at either common or external iliac levels. The major collaterals for the aortic obstructions centered about the rectum and the iliac crest. The inferior mesenteric or the anastomosis from mid- to left colic were the main affluents for the first pathway, the lumbar arteries for the second. The role of the internal mammary-inferior epigastric pathway is uncertain; some of our cases showed filling of the inferior epigastric indicating the artery was not being used as a collateral.

The common iliac obstructions, of which there were 23 cases, tended to show diffuse arteriosclerosis with frequent concomitant occlusion of the external iliac, hypogastric, and femoral systems. The collaterals in the two instances of bilateral common iliac occlusion were similar to those of the aortic cases. In the unilateral closures, the pathways, in order of importance, were: iliac, sacral, transverse, and rectal. The latter were not used except in extremely diffuse disease.

Occlusion of the external iliac often included the common femoral artery. The collaterals were almost always between the descending branches of the hypogastric artery leading to the femoral circumflex and the profunda femoris arteries. The iliac pathway was used only when the terminal part of the external iliac was patent. Concomitant obstruction of the common and external iliacs, which was quite common, brought into additional play rectal, iliac, sacral, and transverse anastomoses.

The occurrence of gangrene could not be related to the height of the obstruction but was somewhat dependent upon closure of vessels usually used as collaterals, and even more upon obstruction of the distributing femoral system of arteries.

It is suggested that in the arteriosclerotic patient obstruction of the inferior mesenteric artery may be responsible for digestive disturbance, obstruction of the sex arteries for diminution in function of the testes or ovaries, and obstruction of the hypogastric artery for neuritis of the lumbosacral plexus.

The anastomoses between the arteries of the various collateral pathways were generally retiform and small. Only the obturator–medial femoral circumflex connection exemplified a bold inosculation with any frequency. The small size of the anastomotic connections correlates with the reduced blood flow which these anastomoses are known to supply.

Since a single collateral pathway is often responsible for the nutrition of the lower limb or limbs, it appears possible for division of one collateral artery to precipitate gangrene.

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EXPERIENCES WITH REPLACEMENT OF SEGMENTS OF DISEASED FEMORAL AND POPLITEAL ARTERIES

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THIS report deals with experiences encountered in 16 instances of replacement of segments of diseased femoral and popliteal arteries. The purpose of the presentation is threefold: (1) to reaffirm the feasibility of replacing segments of diseased peripheral arteries as demonstrated by Julian and his associates,[1] Rob and Eastcott,[2, 4] and Shaw and Wheelock[3]; (2) to point out that it is possible to use plastic cloth tubes successfully in the smaller vessels; and (3) to stress a few fundamental considerations which must be recognized in dealing with these peripheral problems.

Of the 16 instances in which vessel segments were replaced, 8 were carried out for aneurysm, 1 of which was ruptured at the time of surgery, 6 were performed for segmental obliteration, and the remaining 2 for aneurysms in combination with obliteration. The location of the replaced segments is shown in Fig. 1. In 3 instances the common femoral artery was replaced as part of a general replacement of the aorta and iliac arteries. Autogenous vein grafts were used in 5 instances with lengths up to 15 cm. A preserved homograft was used in one case, in a patient with twin femoral aneurysms, while a plastic cloth tube of Vinyon-N was inserted for comparison in the opposite extremity. In the remaining instances the replacements were made with plastic cloth tubes up to 42 cm. in length, composed in 8 instances of Vinyon-N, in one instance of collandered Dacron, and in the remaining instance of nylon A, the latter two substances being presently under investigation in our laboratories by Dr. Ralph A. Deterling, Jr. In all but 4 of the cases where the situation was urgent or obvious, preliminary arteriograms were performed in an effort to ascertain the degree of patency of the peripheral bed. Delays of from 3 to 5 seconds were employed in the arteriograms in an effort to outline the bed distal to the level of block. The aneurysms were resected, except for those portions of the sac juxtaposed to the accompanying vein which were usually left in situ to avoid injury to the vein. In the case of the segmental occlusions no effort was made to remove the obilterated segment, but the artery was divided above and below and the substitute artery was laid alongside the diseased vessel, after anastomosing end-to-end above and below. If good pulses were palpated in the feet following the insertion of the new arterial segment, no anticoagulation treatment was employed. If the oscillometrics and pulsations were questionable, then anticoagulation therapy with heparin and Dicumarol was instituted in the postoperative period. Where the peripheral pulses and the oscillometrics have

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

remained good following segmental replacement, we have made no effort to visualize the area since it seems to us that the clinical signs and symptoms are sufficient. If, however, there has been any question as to the patency of the vessel, we have tried to check this with a repeat arteriogram. The follow-up period ranges from one and one-half months to three years, and our longest follow-up on a plastic cloth prosthesis is ten months. This latter is in a popliteal artery segment and is functioning properly with good peripheral pulses and no symptomatology at this time.

Eleven of the replacements are completely satisfactory with good distal pulsations and oscillometrics. An additional case is of questionable patency, although symptomatically the calf and foot pain has been relieved. Checkup arteriography has not been made in this patient because of recurrent thrombophlebitis in this leg and subsequent pulmonary embolism.

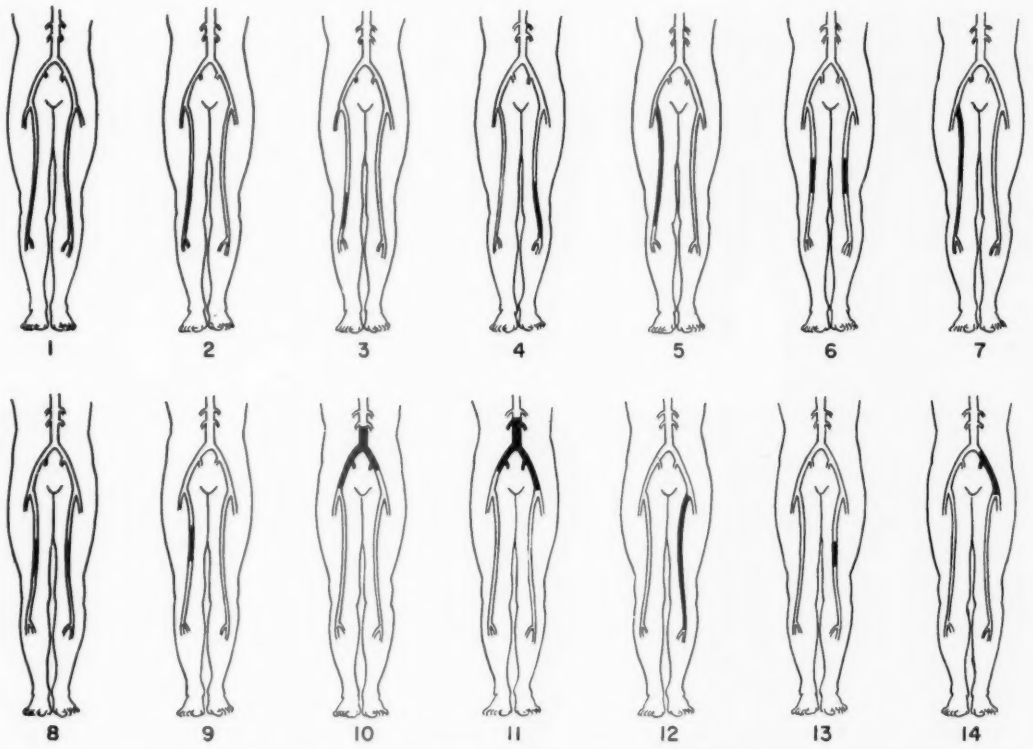

Fig. 1.—The sites of segmental replacement of the femoral and popliteal artery. In three instances the common femoral artery was replaced along with the iliac vessels.

One case of replacement of the left iliac and common femoral vessels with a Vinyon-N cloth prosthesis occluded in the immediate postoperative period, as proved by a subsequent operation. No pulses had ever returned in this patient after insertion of the prosthesis, and re-exploration revealed that the superficial femoral vessel below the profunda level was completely obliterated to the popliteal artery level, and in essence the prosthesis had been anastomosed to a short segment of open vessel with little or no run-off. Another instance of obliteration occurred in a 42 cm. Vinyon-N cloth replacement of the entire superficial femoral and most of the popliteal artery. Immediately following operation no improvement was noted in the oscillometrics and no peripheral pulses were felt. A repeat arteriogram showed that

the prosthesis had become obliterated. Here again it seemed that the prosthesis was anastomosed to a small segment of open vessel with little or no peripheral run-off. One patient with a mycotic aneurysm of the femoral artery, occurring secondary to subacute bacterial endocarditis, died in the early postoperative period of coronary artery embolism. At autopsy, the vein graft was patent. The final unsuccessful outcome occurred in a young woman with bilateral femoral artery aneurysms and malignant hypertension. An autogenous vein graft was employed in replacement of the left femoral artery segment and a Vinyon-N cloth prosthesis was employed in the right leg. Both prostheses were functioning nicely and patient was doing well until three weeks after the second operation when she was placed on a regimen of antihypertensive medication with Apresoline in order to reduce her blood pressure. After two doses of the drug the patient's blood pressure dropped from 220 to 80 mm. Hg systolic and she immediately began to complain of severe pain in the right leg. The entire lower leg became ice cold and obviously ischemic. She was explored within two hours of this episode when it was found that the hypotensive period had resulted in an acute obliteration of the superficial femoral artery well above the level of insertion of the prosthesis at the point of marked narrowing, which had hitherto gone unrecognized. The clot hung down from this level into the prosthesis in a long tail, although the majority of the prosthesis appeared completely free of attachment of thrombus to its wall. The clot was removed and good flow proximally was obtained and a new prosthesis was inserted, but no distal retrograde flow was ever secured. Immediately after releasing the hemostatic clamps the new prosthesis clotted and it seemed apparent from this that there had been seeding of clot into the peripheral bed, so that there no longer was sufficient run-off to allow the vessels to remain open.

Four of the plastic cloth prostheses traversed the popliteal space. In none of these patients does flexion of the leg beyond 90 degrees at the knee cause any reduction in pulse at the foot. The theoretical objection to the employment of cloth prosthesis in this vicinity, therefore, has not held in our experience.

DISCUSSION

While this series is not as large as several that have been reported dealing with peripheral replacements, the combined experience of Drs. Arthur H. Blakemore, Ralph A. Deterling, Jr., and myself at the Presbyterian Hospital numbers over 100 replacements of the aorta and peripheral vessels for aneurysm and segmental occlusion. Over 50 of these replacements have been with tubes of plastic cloth. With this experience as a background, it seems justifiable to make the following points referable to this work.

To begin with, in the segmental occlusions objection is occasionally raised to the concept of resorting to what is considered radical surgery for a process that may be static and well tolerated. It is conceivable that segmental occlusion reaches a static point in a few individuals. However, it is impossible to predict that individual in which obliteration will progress, and if one reviews the history of those who have passed the stage where replacement therapy is possible, it is apparent that the disease process has been a progressive one, punctuated by periods of rather abrupt worsening which may correspond with the pinching off of the major collaterals by advancing clot. Fig. 2 is representative of what we have seen rather frequently in visualization studies. A clot is hanging down from the distal limit of the occlusion, actually at the very mouth of the next major collateral. It is quite apparent that within the very near future this collateral will become plugged and that the thrombus will extend to the level of the next major collateral. Further, the only sure way of completely

eliminating symptoms is by restoration of the circulation. It is seldom that sympathectomies or vasodilator drugs ever completely eliminate claudication, although they may improve the situation tremendously.

Fig. 2.—Arteriogram of the right superficial femoral artery. The proximal segment is occluded and the distal segment is widely patent. However, clot formation is proceeding in the area of entrance of the first major collateral. It is apparent that this collateral will soon become obliterated.

The next point is that in our experience it would seem that the most important single factor in determining the patency of a graft or prosthesis is the run-off into the peripheral bed. If this peripheral bed is widely open, if the flow of blood through the graft or prosthesis is rapid, and if there is a return of pulses in the feet, then patency is usually assured. In such a case,

it would seem to make very little difference whether the gap were bridged with a synthetic plastic, a homograft, or an autogenous vein graft. Even a relatively long prosthesis apparently will remain patent, provided the peripheral bed is adequate. A prosthesis of Vinyon-N measuring 38 to 40 cm. in length, replac-

Fig. 3.—Arteriogram of the left femoral artery showing a blind segment of open vessel in the lower thigh. Anastomosing to such open segments of vessel is a source of failure in peripheral replacements.

ing the entire superficial femoral and most of the popliteal artery, has now been in place for six months, functioning nicely with excellent peripheral pulses and oscillometric readings.

Preoperative evaluation of the peripheral bed is dependent to a considerable extent upon the arteriogram. In this respect, I should like to call your

attention to a pitfall in interpretation which may result in the erroneous belief that a case may be operable. Fig. 3 shows an arteriogram which reveals a blind segment in the superficial femoral artery. The fact that this was a blind segment was revealed only after a picture was taken with a five-second delay following injection. Earlier films showed the artery somewhat hazily in this area which gave the impression that the clot existed only from the bifurcation of the common femoral to approximately the mid-thigh region. Bleeding is not necessarily a sign at operation that the vessel is patent peripherally; as can be seen from this arteriogram, some of these blind segments communicate with a fairly large collateral vessel. If one mistakenly plugs into such a segment, thrombosis is inevitable.

SUMMARY

Segments of the femoral or popliteal arteries have been replaced in 16 instances because of either obliterative disease or aneurysm. In 10 instances this replacement has been achieved with tubes made of synthetic fabric and in 4 instances these tubes have traversed the popliteal space without, apparently, any disturbance in flow during flexion. Thrombosis has occurred in 2 of the prostheses because of inadequate run-off in the peripheral bed, or, in other words, plugging into a blind segment of open vessel. Thrombosis occurred in the third case above the site of replacement following a hypotensive episode. The most important factor in determining patency of the prosthesis would appear to be the capacity of the peripheral bed. In segmental occlusion it would seem impossible to predict which cases may proceed with progressive obliteration. Therefore, the earlier the situation is corrected, the smaller the segment required for replacement, the better the peripheral run-off, and the more likely the chances of success. It would seem desirable to approach the early and minimal claudicators rather aggressively with a view toward determining the presence or absence of a small segmental block which may be favorable for correction.

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THE USE OF VESSEL GRAFTS IN THE TREATMENT OF POPLITEAL ANEURYSMS

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A SATISFACTORY report pertaining to the absolute incidence of aneurysms of the popliteal artery has not been found in the literature. Autopsy material particularly is lacking because of the infrequency with which the popliteal space is included in the post-mortem examination. Clinical studies are heavily loaded with symptomatic aneurysms which were the cause of the patient's seeking medical care. Aneurysms of this region, however, are apparently less common than that of the bifurcation of the aorta and more common than anuerysms within the chest and the major arteries of the upper limbs and neck.

The outstanding etiological factor in the series which have been reported has been arteriosclerosis, although occasional syphilitic and micotic aneurysms are included. Traumatic aneurysms of the popliteal region, as in other regions of the body, are ordinarily false aneurysms or pulsating hematomata.

In an analysis of 100 cases of popliteal aneurysms, Gifford, Hines, and Janes[1] found that 92 of the patients were arteriosclerotic, 3 syphilitic, 1 micotic, and 1 traumatic. An extremely high incidence of complications in this series reflects the fact that most of the aneurysms were present for study because of the complications. The complications included 20 acute thromboses of the aneurysm, rupture of the aneurysm in 16, peripheral emboli arising in the lesion in 14, and gangrene of the extremity in 24 patients. A popliteal aneurysm might well be expected to be prone to complications such as rupture or thrombosis because of the constant motion to which it is subjected. The liability for complications has made the lesion the subject for many forms of surgical treatment.

The most elementary form of surgical treatment was the ligation of the popliteal artery proximal to the dilatation. This operation was undertaken by Hunter who, in the eighteenth century, ligated the femoral artery in the adductor canal. Matas[2] stated that 10.5 per cent of patients operated upon by this method developed gangrene of the extremity. Matas' improvement consisted of this standard endoaneurysmorrhaphy. The particular feature of this operation is that interruption of collaterals is avoided by approaching the vessels entering and leaving the aneurysm through the sac itself, and there is essentially no dissection external to the aneurysm. By thus preserving collateral arterial channels Matas reported an incidence of gangrene of 5.2 per cent in 154 patients

Read at the ninth annual meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 5, 1955.

treated by this method. This operation leaves much of the aneurysmal sac in the popliteal space. Whether or not this is productive of undesirable complications is questionable but, subsequent to Matas' method, aneurysmectomy has been frequently used, obtaining protection against gangrene following total removal by a preliminary lumbar sympathectomy. Linton[3] reported this combination of surgical procedures. Fourteen patients are included in his study. Eleven of these had spontaneous aneurysms on the basis of arteriosclerosis. Gangrene did not develop in any patient so treated and in only one of the operations did the circulation seem precarious for a few days following the aneurysmectomy. In all of the cases operated upon by Linton, the aneurysms pulsated at the time of surgery. The dorsalis pedis pulse was palpable in 4 instances and the posterior tibial in 6. The aneurysm pulsation and that of both arteries at the ankle were present in 2 of the cases operated upon. From these figures it can be determined that of the 14 aneurysms operated upon, 8 had direct pulsatile circulation into the extremities through the aneurysm. Linton did the two operations in separate stages, first the lumbar sympathectomy and then, after a period of nine to ten days, proceeding with the aneurysmectomy. Janes and Ivens[4] have reported the same combinations of surgical procedures. In this series, as well as in Linton's, no legs were lost and there were no deaths. Gifford and associates[5] have added a case treated successfully by this method. This one is of particular interest because the lesion occurred in a 35-year-old patient and was apparently arteriosclerotic. In this instance, a lumbar sympathectomy and extirpation of the aneurysm were performed at the same session with a satisfactory result.

These experiences lead to the conclusion that about a 10 per cent incidence of gangrene is to be expected by the method of proximal ligation of the aneurysm. Improvement of this figure results if the vessels are sutured from within the aneurysm according to the method of Matas, and, if a lumbar sympathectomy is added to the armamentarium, the incidence of gangrene is diminished essentially to zero.

Fundamental to these good results is the fact that an aneurysm of an artery constitutes in itself some degree of arterial obstruction and is effective in the development of collateral arterial pathways. This automatically then produces the same effect as has been sought by some in the past through the use of occlusive clamps applied externally over an artery later to be ligated for the development of collaterals. Final efficiency of the collateral bed is best demonstrated in those cases of popliteal aneurysm which have progressed in size until obstruction has been produced, as is evidenced by the loss of pulses at the ankle. In such patients as this, careful removal of the aneurysm will diminish the mechanical obstruction to the collateral bed by removing the space-occupying lesion from the limited popliteal area. It would seem that more danger of interfering with the circulation of the limb exists in those patients who are operated upon before the pulses at the ankle are lost.

Even though apparent freedom from gangrene is obtained by lumbar sympathectomy preceding extirpation of the popliteal aneurysm, the patient is left with a less than normal circulatory system, and a significant occurrence of

TABLE I

PATIENT	AGE	ETIOLOGY	SYMPTOMS	DURATION	PREOP. PULSES IN ANKLE	TYPE OF SURGERY	SYMPATHECTOMY	RESULT
1. A. K.	30 M	Mycotic; sbe; left	Painful mass	3 wk.	+	Excision and vein graft (auto.)	0	Pulses maintained; patient died 3 days postop. of heart disease
2. A. P.	67 M	Arteriosclerosis; acute thrombosis! right	Pulsating mass; gangrene 3 toes	2 yr. 3 days	–	Excision and vein graft (auto.)	0	Pulses restored; superficial necrosis healed 2 yr.
3. G. P.	39 F	Sclerotic; bilateral; acute rupture	Acute pain; pulsating masses	3 wk.	+ +	Bilateral excision and vein graft (auto.)	Left + Right 0	L.—no pulses; no rest pain; claudication 1½ blocks Rt.—pulses restored; asymptomatic 5 mo.
4. R. L.	38 M	Sclerotic; right	Painful pulsating mass	15 mo.	+	Excision and graft (homo. iliac artery)	0	Pulses maintained; complete recovery 6 mo.
5. J. H.	77 M	Sclerotic; left	Claudication; large tender pulsating mass; superficial necrosis first toe	3 yr. 2 wk.	–	Excision and graft (homo. aortic segment)	0	Posttibial pulse restored; necrosis healed 3 mo.
6. J. S.	73 F	Sclerotic; left	Acute rupture into thigh; femoropopliteal	10 days	+	Excision and graft (auto. vein)	0	Pulses maintained 2 mo.
7. G. H.	76 M	Sclerotic; left	Sudden pain; left calf	1 wk.	–	Excision and graft (homo. vein)	+	Pain relieved; pulses not restored
8. A. L.	57 M	Sclerotic; right	Pulsating mass; mild pain	5 wk.	+	Excision and artery graft (tailored homo. aortic segment)	0	Pulses maintained 10 mo.

such symptoms as intermittent claudication must be expected. The method of implantation of a suitable vessel graft after removal of the aneurysm should re-establish the circulation and prevent such late symptoms. Pringle[6] reported the use of a vein graft to maintain direct arterial circulation after removal of a popliteal aneurysm in 1913. In his case the patient was 49 years of age and the aneurysm had been produced in an arteriosclerotic artery by a minor injury four months prior to the development of the aneurysmal swelling. Removal of the lesion left a defect 2 inches in length which could not be reapproximated even with flexion of the knee. The defect was repaired by implanting a segment of saphenous vein. During the period of observation reported in the paper, the pulses at the ankle remained normal and the patient returned to work. Other instances of restoring continuity of the popliteal artery were reported prior to his case. In volume V of Keen's *Textbook of Surgery*,[7] Matas mentioned that J. B. Murphy of Chicago suggested in 1897 the possibility of restoring continuity of the artery both by lateral and circular arteriorrhaphy after excision of aneurysm or arteriovenous fistula. Enderlen[7] of Wurzburg in 1907 removed a popliteal aneurysm and restored continuity by direct anastomosis. In this case flexion of the knee was required in order to bring the vessel ends together. Lexer[7] reported the implantation of a venous transplant following removal of a traumatic aneurysm of the axillary artery in 1907. Pulses in the arm were restored, but on the fourth day thrombosis of the graft apparently occurred and the arm became gangrenous. Delbet[7] interposed an artery into a long defect produced by removal of a popliteal aneurysm in a 74-year-old man. His operation was performed in 1906 and reported in 1907. The procedure failed completely, however, because the artery used was taken from an amputated limb in which the diagnosis was arteriosclerosis and the pathology in the graft made it unsuitable.

The purpose of the present paper is to report the results obtained in 9 aneurysms in 8 patients with implantation of a vessel graft after complete resection. Data pertaining to these cases are summarized in Table I. One patient had bilateral aneurysms, making a total of 9 lesions operated upon. Eight of these aneurysms were the result of arteriosclerosis and 1 was a micotic aneurysm occurring during the course of an acute subacute bacterial endocarditis.

CLINICAL NOTES

Rather than include detailed histories of the patients shown in Table I, salient features will be reported.

CASE 1.—This young man developed a severely painful pulsating mass in his left popliteal space during the third week of an acute illness characterized by chills, high fever, splenomegaly, and diffuse cutaneous petechiae. The diagnosis of subacute bacterial endocarditis was made and positive blood cultures were obtained. Operation was undertaken because the popliteal aneurysm became adherent to the skin of the popliteal space and, over the course of some days of observation, produced thinning of the skin, threatening to rupture. The pain produced by the lesion was also a surgical indication. Excision and graft with a 3 inch segment of autogenous saphenous vein was carried out, relieving the pain and maintaining the pulses at the patient's ankle. The patient's immediate postoperative course was favorable but he died three days postoperatively of heart failure. At post-mortem examination the popliteal space was included and showed a patent graft.

CASE 2.—This patient was seen because of the acute onset of pain and the development of severe ischemic changes in the first, second, and third toes of the involved extremity. The patient had known of pulsations in a mass behind the right knee for two years prior to his admission. A feeble pulse was palpable in the large aneurysm that was found on examination, but pulsations at the ankle were absent and the oscillometric index was less than one

A. *B.*

Fig. 1.—*A*, Arteriogram of right femoral-popliteal system in Case 3. The aneurysmal sac is visualized as is the popliteal artery distal to the lesion.

B, Femoral arteriogram of left leg in Case 3. The aneurysm is not visualized but patent artery is seen distal to the lesion.

until below the knee. Excision of the aneurysm was followed by the implantation of an autogenous saphenous vein graft. The pulses at the ankle level were restored completely. Following surgery the first, second, and third toes showed a continuation of the development of gangrene of the skin, but during the second week following surgery the black superficial layers and the toenails desquamated, leaving viable skin beneath. The nails regenerated and the patient's foot had a normal appearance at the time of discharge. The leg has remained entirely asymptomatic during the twenty-six-month period since surgery.

CASE 3.—This patient is unusual in that bilateral arteriosclerotic aneurysms developed at the age of 39 and also because rupture occurred in both aneurysms almost simultaneously. The patient complained of an acute, painful mass occurring first behind the left knee, and

Fig. 2.

Fig. 3.

Fig. 2.—Exposed aneurysm in Case 4. The nerve trunk and popliteal vein have been freed from the lesion.

Fig. 3.—The aneurysm has been resected and an iliac artery homograft implanted in Case 4.

then, within twenty-four hours, behind the right. Pulses were palpable at the ankle on both sides but were very weak on the right as compared with the left. Arteriograms were made on both sides. That on the right (Fig. 1, *A*) shows a saccular aneurysm very much

smaller than would be suspected on the basis of the size of the pulsating mass. The aneurysm almost completely occluded the artery but some dye is visible in the artery distal to the lesion. On the left side the arteriogram (Fig. 1, *B*) was taken in a direct posteroanterior position. Numerous collateral vessels are seen, but no dye is present within the area of the lesion or it is obscured by the lower end of the femur which overlays the aneurysm. Dye is present in the popliteal artery distal to the aneurysm. Bilateral excisions and autogenous saphenous vein grafts were carried out simultaneously. Considerable difficulty was experienced on the left side in producing a satisfactory distal anastomosis between graft and artery because of the severe changes in the popliteal artery at this point. Pulses were restored completely on the right side but no pulses were present on the left following excision. There was no sign of ischemia of the left foot at rest, but when the patient was allowed complete ambulation severe claudication occurred after one and one-half blocks of walking. Consequently, a lumbar sympathectomy was performed on that side. It should be noted that the sympathectomy did not produce an increased ability to walk.

CASE 4.—This patient is also of a young age group for development of an arteriosclerotic aneurysm. His symptoms had been present for fifteen months prior to surgery. They consisted only of the knowledge of an occasionally tender, pulsating mass behind the knee. At surgery the aneurysm was found to be of moderate size and the artery above and below was comparatively normal (Fig. 2). Excision was followed by the implantation of a frozen homologous iliac arterial segment (Fig. 3). This level of homologous graft was used because it perfectly matched in diameter the artery above and below the lesion. No sympathectomy was performed. Pulses were maintained following surgery and complete recovery ensued.

CASE 5.—This patient complained of claudication in the involved extremity for a period of three years before surgery. He had knowledge of the presence of a pulsating mass during most of this time. Two weeks before surgery a gangrenous ulcer appeared on the tip of the first toe. Excision of the aneurysm, which was the largest in diameter in this series (Fig. 4), was followed by implantation of an aortic segment. The large diameter of the artery in Hunter's canal above the aneurysm necessitated use of this size graft. The vessel was smaller distally and the defect was fitted perfectly by using the aortic bifurcation, anastomosing the aorta end proximally and one of the common iliac arteries distally (Fig. 5). The other iliac artery was closed flush at the bifurcation with a double line of sutures. The pulses at the ankle were absent before surgery but they were found to be restored after operation. The gangrenous ulcer of the toe slowly healed.

CASE 6.—This case is that of a 73-year-old woman who had had an acute onset of pain and swelling of the left lower extremity ten days prior to surgery. The initial clinical impression was acute thrombophlebitis and the patient was put on anticoagulants. The enlargement of the thigh, however, became more and more severe, and signs of blood loss became manifest. A bruit was heard over the course of the femoral vessels in the thigh from groin to knee and at this time vascular consultation was sought. The swelling was found to occupy the popliteal space and much of the thigh, showing the pulsations of a false aneurysm. Surgery was undertaken under the preoperative diagnosis of a rupture at some point in the femoral arterial system without, however, indication as to its exact localization. Proximal control of the femoral artery was obtained high in the groin after which the hematoma was entered and explored. An aneurysm of the femoral-popliteal junction at the level of the adductor canal was found. The aneurysm was localized to a 3 cm. segment of artery and was 5 cm. in diameter. Rupture had occurred through the proximal end of the aneurysm. The majority of the blood loss had occurred upward into the compartments of the thigh. Resection of the involved segment of artery was followed by implantation of a 4 cm. segment of the patient's saphenous vein. The pulses at the ankle, which were present before surgery, were maintained, and over a period of some weeks the thigh became essentially normal.

CASE 7.—This patient is a 76-year-old man whose onset of symptoms was sudden, occurring two to three days before admission to the hospital. Pulses at the patient's ankle were

not palpable on admission, but the aneurysm pulsated vigorously. A lumbar sympathectomy was performed seven days prior to excision and graft. The aneurysm was found to be the longest one in this series, extending from a point high in Hunter's canal down to the bifurcation of the popliteal artery. Because of elongation the aneurysm was curved outward and laterally in the popliteal space. This elongation had caused acute angulation of the

Fig. 4.

Fig. 5.

Fig. 4.—Large popliteal aneurysm in Case 5. Attempt was made to preserve the vein but was finally abandoned because of adherence to the aneurysm.

Fig. 5.—Aortic bifurcation implanted with aorta end at proximal end on the left and a common iliac attached distally on the right. The sutures closing the other iliac stump are visible.

anterior tibial artery occluding it at its point of perforation through the interosseus membrane. The angulation brought about by the fixed point of perforation of the anterior tibial artery had produced obstruction of the posterior tibial artery. Retrograde bleeding, however, was obtained from both of these vessels when the distal end of the aneurysm was

divided. A fresh homologous saphenous vein graft was used to bridge the defect from above the adductor hiatus to the bifurcation, and although pulses did not reappear at the ankle the graft continues to pulsate three months following surgery, and the nutrition of the foot is good. The lack of pulsations at the ankle indicates that the major branches of the popliteal artery were occluded by arteriosclerotic changes and that the graft itself must transmit blood downward to collateral vessels below the knee.

CASE 8.—This patient's aneurysm produced only moderate pain which became more severe during any exercise. Excision produced a defect of 7 cm. which was restored using a frozen homologous iliac segment. In order to match the nearly normal popliteal artery diameters above and below the resected lesion, the iliac graft was narrowed by cutting out a longitudinal strip. The graft was restored to a cylinder with a longitudinal everting mattress suture reinforced with a continuous over-and-over suture in the everted edges. The pulses at the ankle have been maintained for ten months since surgery.

COMMENTS

During the operations on these patients certain observations seemed to be made consistently. It was noticed in all of the large aneurysms that the factor of elongation in the stretched vessel tended to angulate the anterior tibial artery

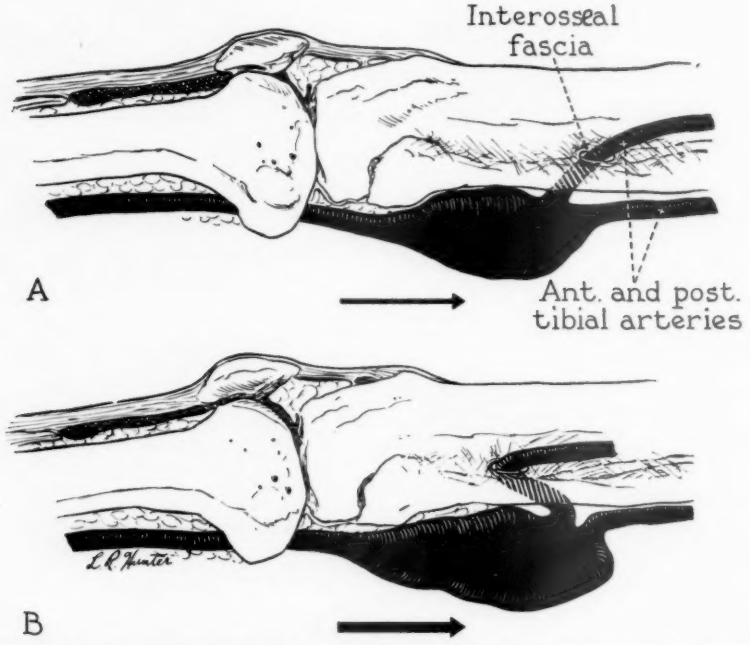

Fig. 6.—Schematic drawing showing tendency for elongation of aneurysm to produce obstruction of both branches of popliteal artery.

(Fig. 6). This should be considered one of the methods by which the pulsatile flow to the ankle is obstructed. In addition, a concomitant obstructive arteriosclerotic lesion may be present distal to the aneurysm, as was assumed in Case 7. A second consistent observation was the infrequency with which open arteries branched from the aneurysm itself. Such arteries as the medial and lateral superior geniculate were either occluded in the wall of the aneurysm or were very small. After obtaining proximal control of the artery above the aneurysm it was possible to open the aneurysm and, after evacuating clots, obtain control

of these vessels flush with the wall of the aneurysm externally. Since they were small or occluded, it did not seem important that they be subjected to special handling.

The majority of the patients were placed on the operating table on their abdomen with the hip opposite the leg to be operated upon elevated on a sandbag about 4 inches in thickness (Fig. 7). The affected leg was kept straight while the opposite one was flexed at the hip and knee. This gave convenient approach to the entire popliteal area and the posterior medial aspect of the thigh above the knee. In this position a flat, s-shaped incision could be made. The upper portion of the incision paralleled the tendons of the medial hamstring muscles. The middle portion of the incision was brought transversely across the popliteal space to about its mid-point and the inferior limb was directed longitudinally down the middle of the calf. This incision avoided the longitudinal incision traversing the knee joint, although Linton[3] and others have used this without apparent disadvantage. The medial placement of the upper portion of the incision aided considerably in the 2 patients in which it was necessary to go above the adductor hiatus to obtain proximal control of the

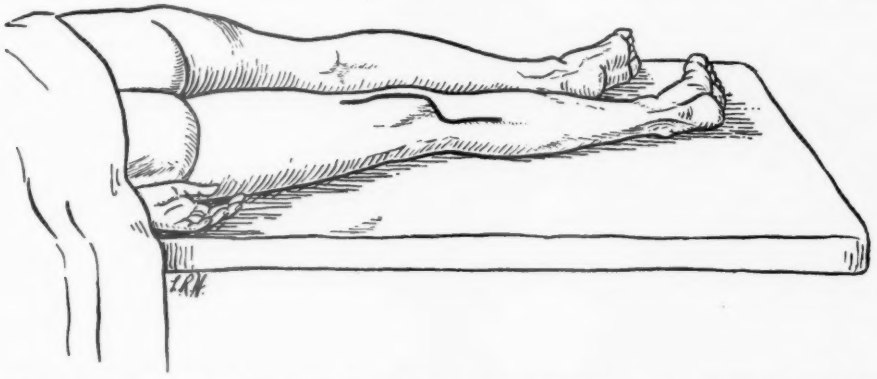

Fig. 7.—Position in which the popliteal space and lower Hunter's canal may be exposed. This was used in the patients of this series, except in Cases 3 and 6.

artery in a more or less normal condition. One of the patients was operated upon in a supine position. This was Case 6 in which the position of the aneurysm was undetermined before surgery and it was thought that it actually was a lesion of the superficial femoral artery. The patient having bilateral popliteal aneurysms in which surgery was performed on both sides simultaneously was operated upon the flat prone position with sandbags under the hips and shoulders to permit good respiratory movements.

By choice, wherever possible, a frozen homologous artery graft was used. The iliac portion of such a graft was used once, while in another case it was necessary to use the abdominal aorta of the graft at the upper end because of the very large size of the superficial femoral artery in Hunter's canal. Vein grafts were used when it was inconvenient to use an artery or when an artery was not available. There has been no observable difference in this period of observation in the late behavior of the two types of grafts. Compared with the superficial femoral artery, the popliteal artery has little muscle compartment

support, and for this reason possible dilatation of the unsupported vein graft in the popliteal space may be looked for. One of the patients (Case 2) has been followed for two years and no change in the vein graft has been noted. It seems that the same lasting qualities of vein grafts in the femoral region may be obtained here.[8, 9]

SUMMARY

1. Nine popliteal aneurysms subjected to excision and restoration through the use of a vessel graft are reported.

2. Five autogenous saphenous grafts, 1 homologous vein graft, and 3 frozen homologous artery grafts were used.

3. One failure to preserve distal artery pulses is reported without serious results other than significant claudication. In this patient a lumbar sympathectomy failed to relieve this symptom.

4. A second patient in whom sympathectomy was performed is included. This preceded the aneurysmectomy and graft. The remaining 7 lesions were removed and grafted without lumbar sympathectomy.

5. In 7 patients the pulses at the ankle were either restored or maintained. Two operations resulted in healing of gangrenous area on the toes.

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(Society for Vascular Surgery articles will be concluded in the December, 1955, JOURNAL.)

Erratum

In the article entitled "A Note on the Incidence and Possible Significance of Valves in the Internal Jugular Vein," by D. L. Sutherland, C. R. Barber, and Eldridge Campbell, which appeared in the August issue of the JOURNAL, pages 320-322, the legend for Fig. 2 on page 321 should be concluded as follows: *S.C.V.*, Subclavian vein.